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THE EFFECT OF HORMONAL TREATMENT ON THE GUMS AND ORAL MUCOSA OF WOMEN

(A) With the Use of Estrogen[1]; (B) With the Use of the Gonadotropic Hormone of Pregnancy Urine[2,3]

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In two recent papers (1, 2), the writer and Blackberg reported observations on the gums of pregnant women and experiments with normal and castrated female rhesus monkeys. "Pregnancy gingivitis" (by us termed "hormonal gingivitis") was found in most of the pregnant women examined and the significant change in these gums was hyperplasia of the epithelium with progressive loss of the keratin layer as pregnancy advanced from the fourth to the ninth month. Similar pathosis was induced in the gums of normal rhesus monkeys injected with pregnancy urine extract. In the second study, with castrate monkeys, Folluetin injections resulted in a condition resembling Vincent's infection, whereas the gums of the estrogen-treated group became hard, pink, lost their pre-existing proneness to easy bleeding, and appeared better able to resist mouth infections.

The present report concerns itself with six patients from Dr. Raphael Kurzrok's clinic for gynecological diseases, three of whom were being injected with estrogen (Progynon B—Schering Corporation) for amenorrhea and three with Folluetin (Squibb) for prolonged menstrual bleeding. Pre-treatment oral observations were not made, first examinations of gums being made two weeks to fifteen months after injections were begun. After gross observation of the gums, a

[1] Estrogen—The material used is the monobenzoate of estradiol commercially known as "Progynon B," manufactured by Schering Corporation.

[2] Extract of pregnancy urine—"Folluetin"—gonadotropic in action, commercially prepared by Squibb.

[3] Presented at the American Association for the Advancement of Science, Section on Dentistry, December 28th, 1936, and International Association for Dental Research, New York Section, January 28th, 1937.

section of tissue was excised, fixed in Bouin's Solution, cut longitudinally from labial to lingual surface and embedded in paraffin with the cut surface down. The tissue was stained with hematoxylin and eosin, and Masson's trichrome. Protocols of individual studies follow:

Case I. Y. A.: Female—age 18 years. Diagnosis: Secondary amenorrhea, due to decrease in estrogenic substance. Treatment: Progynon B—20,000 rat units during two weeks prior to mouth examination and biopsy. .3 g Thyroid t.i.d. concurrent with Progynon B. *Gross Examination of Gums—3/31/36:* Slight marginal gingivitis on lingual surfaces of lower bicuspids and molars. Slight edema of crests of interdental papillae of lower anterior teeth; otherwise mouth negative. *Microscopic Findings:* Biopsy: Buccal interdental papilla distal to upper right second bicuspid (negative to gross appearance). Alveolar gingivae (the term "alveolar gingivae" is used to denote section of pink gum covering alveolar process): surface keratin well marked; granulosa in a few areas; few large and many small epithelial pearls; prickle cell layer compact with good intercellular bridges and hyperplastic; slight chronic inflammatory reaction. Areolar gingivae (the term "areolar gingivae" denotes the mucous membrane covering bone beyond the alveolar process): small amount of surface keratin; no granulosa; no pearls; prickle cell layer: loose tissue with few prickle cells and hyperplastic; chronic inflammatory reaction and slight edema. In both, vascularity in papillary layer and denseness of collagen in subepithelial layer not grossly altered.

Case II. B. H.: Female—age 31 years. Diagnosis: Amenorrhea. Treatment: Progynon B—85,000 rat units during eight weeks prior to mouth examination and biopsy. *Gross Examination of Gums:* The gingivae of normal color in some section; cyanotic, swollen, glossy in others. General recession throughout mouth, more marked in region of lower right central incisor and lower left first bicuspid. Evidence of non-supperative periodontoclasia with heavy tartar formation. Marginal inflammation on lingual of lowers and labial and buccal of uppers. Some recession of crests of papillae throughout. Despite evidence of pre-existing pathosis, gums firm without tendency to easy bleeding on traumatization. *Microscopic Findings:* Biopsy: upper right second bicuspid edentulous area (negative to gross examination). Alveolar gingivae: surface keratin well marked; no curls; granulosa seen; few large and several small pearls; prickle cell layer of stratum germinativum well seen and hyperplastic (*fig. 8*). Areolar gingivae: some surface keratin; curls seen; no granulosa; no pearls; prickle cell layer compact and flattened near surface and hyperplastic (*fig. 12*). In both, a few small foci of lymphocytes in subepithelial layer; vascularity not grossly altered.

Case III. L. S.: Female—age 31 years. Diagnosis: Secondary amenorrhea due to lack of estrogenic substance. No period for past two years. Treatment: Progynon B—160,000 rat units during three months prior to mouth examination and biopsy. *Gross Examination of Gums:* Gums on whole pink, slightly thickened, hard and stippled. Slight marginal inflammation in regions of lingual lower molars, buccal lower third molars and buccal upper right first molar. Lower

anterior papillae slightly edematous, old rose color crest, no evidence of inflammation. Mouth clean and healthy with no tendency to hemorrhage. *Microscopic Findings:* Biopsy: upper right second bicuspid edentulous space (negative to gross appearance). Alveolar gingivae: surface keratin well marked near tip; no curls; granulosa well developed in some parts; pearls; prickle cell layer well seen and hyperplastic; slight inflammatory reaction in subepithelial layer (*figs. 9 and 10*). Areolar gingivae: definite keratin layer seen in places; some curls; no granulosa; no pearls; prickle cell layer in few places and hyperplastic; marked inflammatory reaction in sub-epithelial layer. In both, vascularity not grossly altered.

Case IV. E. C.: Female—age 11 years. Diagnosis: Puberty bleeding continuous five weeks prior to injections. Treatment: Folluetin—3,200 rat units during three weeks prior to mouth examination and biopsy. *Gross Examination of Gums:* Debris along gingival margins throughout, more marked on left side in molar region. In general, gingivae pale pink. Bluish discoloration in upper anterior region. Mucous membranes pale. General thickening of marginal gingivae but no redness. Interdental papillae in regions of upper and lower bicuspids and molars slightly swollen. *Microscopic Findings:* Biopsy: Interdental papilla distal to upper right first bicuspid (slightly swollen). Alveolar gingivae: keratin well marked; no curls nor pearls; well developed granulosa; prickle cell layer well seen (not as thick as in Progynon B cases); heavy inflammatory reaction at tip and increased vascularity. Areolar gingivae: no keratin; no curls nor pearls; no granulosa; prickle cells not seen; slight inflammatory reaction with increased vascularity; pyknotic neuclei in prickle cell layer.

Case V. G. C.: Female—age 30 years. Diagnosis: Sterility function due to decrease of estrogenic substance. Polyp of uterus. Hyperplasia of endometrium. Menstrual bleeding four days each week. Treatment: Approximately 25,000 rat units of Folluetin during fifteen months prior to mouth examination and biopsy. Similar treatment using similar hormones of other manufacturers for about one year previous. *Gross Examination of Gums:* Marginal inflammation general. Lower anterior papillae chronically inflamed, of soft texture, bleeding easily on traumatization. Calculus present. Upper gingivae pale, not as inflamed as lower. Pseudo-papillae seen in regions of upper lateral and upper and lower bicuspids, six papillae being thus affected. Slight swelling and blunting of papillae in bicuspid regions. *Microscopic Findings:* Biopsy: interdental papilla distal to upper right first bicuspid (slightly swollen). Alveolar gingivae: surface keratin near tip; no curls; granulosa seen; no pearls; prickle cell layer not as thick as in Progynon B group and pegs more pointed (*fig. 5*). Areolar gingivae: some keratin in places on surface of heavy layer of parakeratosis; one curl seen; no granulosa; no pearls; no prickle cells seen. In both, slight evidence of inflammatory reaction, questionable increased vascularity and edema in papillary layer. Cheek mucous membrane; surface keratin not seen. Prickle cells never well formed. Many cells vacuolated and pale; definite evidence of hydropic change. No evidence of inflammation (*fig. 4*).

Case VI. D. S.: Female—age 15 years. Diagnosis: Hyperplasia of endometrium. Profuse bleeding for one and a half months. Treatment: Prolan—2400 rat units during seven weeks prior to biopsy. 1600 rat units Prolan during nine weeks following. (Prolan is the gonadotropic hormone of pregnancy urine, similar to Folluetin.) *Gross Examination of Gums (3/16/33) and Dental History:* Patient receiving specific treatment in Vanderbilt Clinic since December, 1929, for congenital lues. Referred to the Dental Department in November, 1930. Examination showed gums normal. Received continuous treatment in dentistry for cavities until January 4th, 1932. Prophylaxis on that date. On January 4th, 1933, patient returned with toothache in lower left first molar. Tooth extracted. Patient was referred to Dr. Kurzrok's clinic on January 24th, 1933, for continuous vaginal bleeding since December 19th, 1932. Treatment began January 25th, 1933, and continued through May 23rd, 1933. Effectiveness of treatment noted the end of January, 1933. In February, patient reported a normal period. Prolan injections continued. On March 16th, 1933, patient returned for treatment of gums (*fig. 7*). History disclosed that during this month she first noticed hemorrhage of gums on brushing teeth; also blisters on palatal and labial sides. Gums somewhat painful. Vincent's smear found positive. Clinical diagnosis: Vincent's Infection. Local therapy for Vincent's begun March 16th. Discontinued March 30th (gums having improved) for observation of further effect of systemic therapy. On examination April 18th, practically negative. Examination April 25th, some marginal inflammation and interproximal gingivitis. On May 23rd, gum papillae of upper right lateral, cuspid, and bicuspid, the upper left second bicuspid and first molar, inflamed and old rose color. Crests of upper right central and lateral, and upper left central and lateral papillae, showed slight proliferation. Lower anterior papillae slightly swollen. Papillae on lingual of upper anteriors on right side swollen and cyanotic with slight proliferation at the crests. Patient noticed hemorrhage of gums on brushing teeth one week prior to date of expected menstruation. Mouth examinations at intervals after discontinuance of Prolan injections (May 23rd, 1933) revealed gums returning to normal without dental aid. Examination on July 3rd, 1933, and subsequently, showed gums entirely normal. *Microscopic Findings:* Biopsy: alveolar gingiva papilla upper left central and lateral incisors, 3/16/33 (*fig. 6*). Keratin at tip not seen. Very poor higher up. No granulosa. No pearls nor curls. Faint staining, ulceration, and evidence of necrosis near tip. In better tissue higher up, prickle cells well formed and compact. Evidence of intense inflammatory reaction with lymphocytes, monocytes, and some recognizable polymorphoneuclear leucocytes present.

DISCUSSION

The changes in the gums of the six women cited were similar both grossly and microscopically to those occuring in the female rhesus monkeys referred to earlier in this paper. Grossly, the gums of the

group receiving Progynon B were found to be in a healthy state generally—firm and not subject to easy bleeding on traumatization—altho varying degrees of slight inflammation were noted. Examinations of Progynon B treated women not included in this discussion, confirm these findings. The significance here does not lie in the mere observation of the condition of the gums because, obviously, an equally healthy appearance of the gums may be found in numerous women not receiving the Progynon B. Rather is it seen in a comparative evaluation of the two groups—Progynon B and Folluetin—the latter of which presented swelling and inflammation of the gums sufficiently characteristic to set them apart. Furthermore, importance must be attached to the similarity of findings in the Progynon B group and in the monkeys (pre-experimentally observed as well) receiving like treatment. The microscopic changes constitute a third factor and add weight to these observations. As previously stated, the Folluetin, treated group showed swelling and inflammation of a typical nature. In addition, one of the cases presented a Vincent's infection. Here again, the significance of the latter finding is open to question when we consider that Vincent's occurs in both men and women not subjected to this hormonal therapy. However, it will be remembered that this particular patient was under dental observation at intervals for approximately two years. During this time there was no evidence of gum pathology until Prolan injections (lasting sixteen weeks) were introduced. Moreover, the gums returned to normal after the injections were discontinued. Hence, there seems to be basis for viewing this history in the light of a possible predisposing relationship. Such an hypothesis is sustained in recalling to mind that in the experiments with castrated monkeys, of the many observed, only three received Folluetin injections. Of these, two developed Vincent's infection. The third died early in treatment. Microscopic findings in the Folluetin group further substantiate the changes cited.

Microscopically, there were marked variations in the two groups (Table I.) Progynon B biopsies showed the keratin layer in the alveolar gingivae to be more definite and firmer (that is, did not strip off as easily in sectioning) than the Folluetin cases. In the areolar gingivae, keratin and curls were seen in all of the Progynon B treated group, while these appeared in only one of the Folluetin group.

TABLE I

Microscopic findings after hormone injections

CASE	AGE	TREATMENT	PAPILLA	STRATUM CORNEUM					STRATUM GRANULOSUM		STRATUM GERMINATIVUM								SUB-EPITHELIAL			
				Thickness		Curls		Parakeratosis	Granulosa		Pearls		Compactness of prickle cells		Thickness of prickle cell layer		Mitosis in single section		Lymphocytes, plasma cells, distorted nuclei, unrecognizable		Vascularity	
				A	A′	A	A′	A′	A	A′	A	A′	A	A′	A	A′	A	A′	A	A′	A	A′
1	18	Progynon B 2 Weeks 20,000 R.U.	|5–6	++	±	0	±	0	±	0	++	0	+	±	++	++	3	0	±	±	+	+
2	31	Progynon B 8 Weeks 85,000 R.U.	|5 Edentulous Space	++	±	0	+	+	+	0	++	0	+	+	++	++	0	5	±	±	+	+
3	31	Progynon B 3 Months 160,000 R.U.	5| Edentulous space	++	+	0	+	+	++	0	++	0	+	±	++	++	2	2	±	++	+	+
4	11	Folluetin 3 Weeks 3,200 R.U.	5–4|	+	0	0	0	0	++	0	0	0	+	0	+	±	0	5	+++	±	++	++
5	30	Folluetin 15 Months 25,000 R.U.	5–4|	+	±	0	±	+	+	0	0	0	+	0	+	±	2	3	±	0	++	+
6	15	Prolan B 7 Weeks 24 cc.	|1–2	± to 0		0			0		0		±		±		2		++++ Acute and Chronic		+	

A = Alveolar Gingivae. A′ = Areolar Gingivae.
±, +, ++, +++, ++++ = Degree of Change. 0 = Absent.

(Keratin is not normally found here.) Pearls were observed in the alveolar gingivae of the Progynon B group whereas none were found in the Folluetin group. In the same gingivae the prickle cell layer was thicker in the Progynon B group than in the Folluetin group. Biopsy of the areolar gingivae showed prickle cells, altho less definite, in the Progynon B group, but none in the Folluetin group. Instead, the tissue appeared loose. This layer was also thicker in the Progynon B group. Slight inflammation was found in the alveolar gingivae at tip of the Progynon B group and of one of the Folluetin group. In the other two of the latter there was marked inflammation at the tip. Both groups showed evidence of some inflammations in the areolar gingivae. An hypothetical explanation of the presence of inflammatory reaction at the tip may be set forth on the ground of local mouth infection via the gingival crevice. However, this does not hold in those cases where the gums were excised in an edentulous space, nor in cases of the areolar gingivae, the latter having no connection with the gingival crevice. It is of interest to note, that the inflamed areas were distant from each other (at tip of alveolar gingivae and in areolar gingivae) with the intervening tissue free of inflammatory reaction. The writer can offer no valid explanation.

Mitosis was observed in all cases, but not sufficiently to prove significant. Vascularity seemed to be slightly increased in some places in the Folluetin group and of questionable increase in the Progynon B group. The changes were not gross enough to be significant. Nor were the changes in the collagen of sufficient intensity to be significant.

In comparing the foregoing microscopic changes with those formerly reported in monkeys we find that, with few exceptions, they are similar in character. The degree of change varied in the monkeys as in the women probably due to the varying length and intensity of treatment. However, the keratin in the areolar gingivae of the Progynon B treated monkeys was thicker, uniformly covering the surface, and inflammatory reactions were not seen. Castrate monkeys receiving Folluetin manifested more uniform changes than the human group. Keratin was consistently poor on the alveolar gingivae. In the areolar gingivae keratin and curls were not seen, and the tendency to degenerative change was more marked and more constant in the monkeys.

A résumé of the changes induced by the gonadotropic hormone of pregnancy urine extract (PU, Prolan, Folluetin) in the four groups studied discloses the following: Hormonal gingivitis ("pregnancy gingivitis") in most cases of the first group (pregnant women) where the hormone is systemic; similar changes in the second group (monkeys, not castrated); changes resembling Vincent's in the third group (monkeys, castrated); and both hormonal gingivitis and Vincent's in the fourth group (humans, non-pregnant and non-castrated). In view of its consistently degenerative reaction on the castrate as well as non-castrate groups, the writer is inclined to the tentative theory that this hormone acts directly on this ectodermal structure.

CONCLUSIONS

1. The hormones Progynon B and Folluetin injected respectively into women in the treatment of amenorrhea and prolonged menstrual bleeding, affect the gingivae and oral mucous membranes. 2. Progynon B injections cause hyper-keratinization and hyperplasia of the oral epithelium. 3. The gums of women receiving Progynon B are uniformly in a healthier state than those receiving Folluetin. 4. The gums of women receiving Folluetin show evidence of inflammation and degeneration both grossly and microscopically. 5. The Vincent's infection reported in one case was probably predisposed by hormonal treatment. 6. Folluetin probably exerts its influence directly on the gums and oral mucous membranes.

The writer wishes to acknowledge his indebtedness to Dr. Raphael Kurzrok, Dr. Philip E. Smith and to Dr. Russell L. Holman of the College of Physicians and Surgeons, Columbia University, and to Dr. Kenneth F. Chase, School of Dental and Oral Surgery, Columbia University, for assistance in obtaining biopsy material.

REFERENCES TO THE LITERATURE

(1) Ziskin, D. E.; Blackberg, S. N.; Stout, A. P.: *Surg., Gyn. and Obstet.*, 57: 719, 1933.
(2) Ziskin, D. E.; Blackberg, S. N.; and Slanetz, C. A.: *J. Den. Res.*, 15: 407, 1936.

Fig. 1

Fig. 2

Fig. 1. Pre-experimental human alveolar gingiva; section from upper left second bicuspid distal papilla. Female, age 26 years. 150 ×. (Orig.)

Fig. 2. Pre-experimental human areolar gingiva; section from upper left second bicuspid distal papilla near mucobuccal fold. Female, age 26 years. 160 ×. (Orig.)

Fig. 3

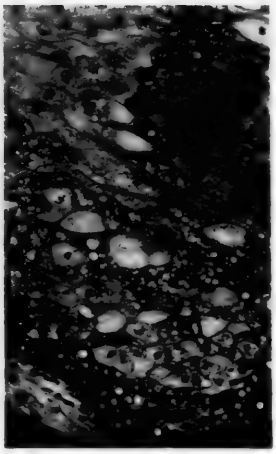

Fig. 4

Fig. 5

Fig. 3. Pre-experimental human cheek mucous membrane. Female, age 22 years. 400 ×. (Orig.)

Fig. 4. Experimental; after 25,000 rat units Folluetin during 15 months. Section from cheek mucous membrane shows hydropic degeneration. Human, female, age 30 years. 400×. (Orig.)

Fig. 5. Experimental human alveolar gingiva; section from upper left first bicuspid distal papilla showing crest. Female, age 30 years. After 25,000 rat units Folluetin during 15 months. 140 ×. (Orig.) Shows poor keratin layer, change in prickle cell layer. Very little inflammatory reaction in subepithelial layer.

Fig. 6

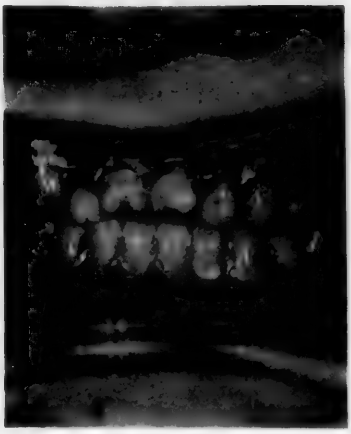

Fig. 7

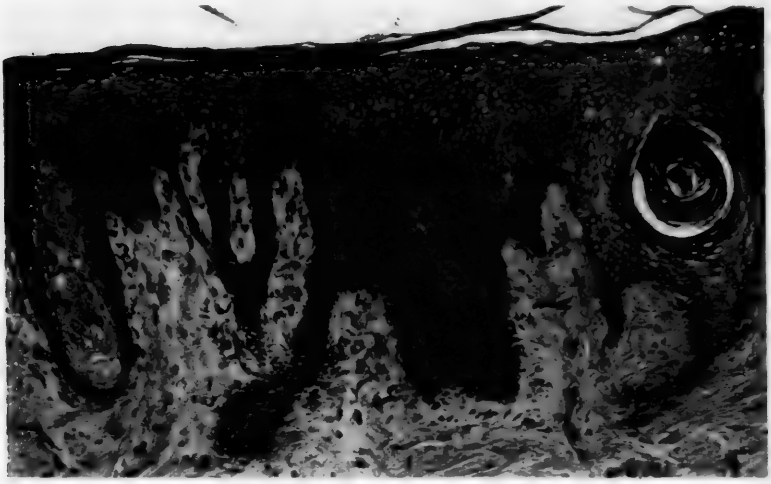

Fig. 8

Fig. 6. Experimental human alveolar gingiva; section from upper left central distal papilla near crest. Female, age 15 years. After 2400 rat units of Prolan for 7 weeks: 117 ×. (Orig.) Shows destruction of keratin and ulceration of surface. Degenerated epithelium with intense inflammatory reaction. Acute Vincent's infection.

Fig. 7. Photograph of gingivae described in *fig. 6.*

Fig. 8. Experimental human alveolar gingiva; section from upper right second bicuspid edentulous space. Female, age 31 years. After 85,000 rat units Progynon B in 8 weeks. 160 ×. (Orig.) Shows good keratin layer; hyperplasia; pearl formation.

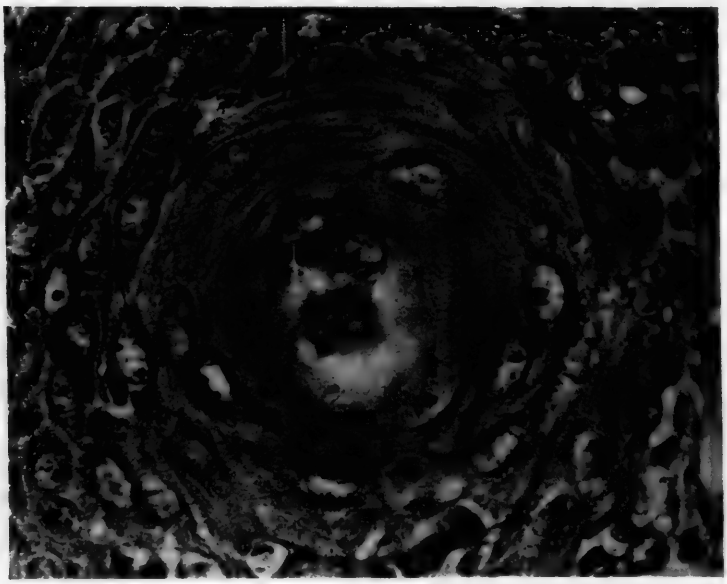

Fig. 9

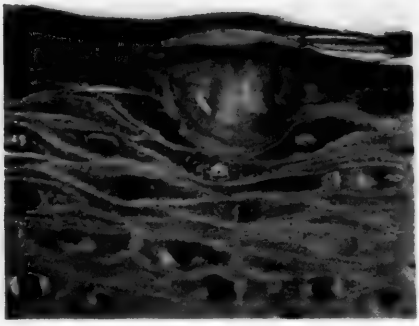

Fig. 10

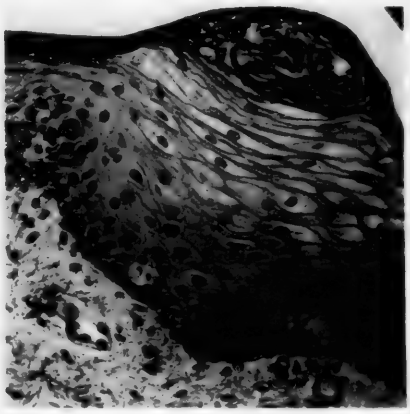

Fig. 11

Fig. 9. Experimental human alveolar gingiva; section from upper right second bicuspid edentulous space. Female, age 31 years. After 160,000 rat units Progynon B in 3 months. 1050 ×. (Orig.) Shows pearl. Granulosa surrounding keratin in center.

Fig. 10. Experimental. Human alveolar gingiva. Section from upper right second bicuspid edentulous space. Female, age 31 years. After 160,000 rat units Progynon B in 3 months. 1050 ×. (Orig.) Shows heavy keratin and pearl underneath pushing down granulosa.

Fig. 11. Experimental human areolar gingiva. Section from upper right second bicuspid edentulous space near mucobuccal fold. Female, age 31 years. After 85,000 rat units Progynon B in 8 weeks. 400 ×. (Orig.) Shows parakeratosis and curl formation.

MORPHOLOGY OF THE FUSIFORM BACILLUS[1]

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F. Vicentini in 1888 (1) made the claim that all "cocci, dumbbell, snake like, spindle-like and other bacillary forms" found in the mouth were propagated by his newly found *Leptothrix racemosa* (*figs. 1 and 2*). Unfortunately Vicentini's sweeping claims tended to discredit his discovery. However, H. Mummery, L. Williams, K. Goadby, and others added valuable, though partly contradictory, contributions to our knowledge of this important organism.

During this study of Vicentini's leptothrix by the author in 1906 (2), and 1908 (3), it was noted that more than one organism exhibited the alga-like dissemination of conida or spores. Because of the scythe-shaped conida shown by this new form, as differentiated from the cocci and rectangular spore type of the *Leptothrix racemosa*, the name *Leptothrix falciformis* (*figs. 3 and 4*) was applied to it. Photomicrographs exhibiting these complex forms have been published by Aisenberg (4), R. D. and M. Dean (5) and E. Coolidge (6). In a previous publication (7), it is recorded that falciform spore bodies or conida are borne on a stem, and that these conida bear a peritrichal flagellation. This feature indicates possession of the power of motility by this organism. Comparison of these spore bodies with the sickle-shaped and fusiform bacilli found intermingled with other organisms of the "materia alba" led to the conviction that the two are identical.

Notwithstanding the use of a variety of culture media, the author's attempts to cultivate these forms have been unsuccessful. Organisms that accurately resemble those found in the mouth never appeared in culture. While engaged in these attempts, Lewkowicz (8) reported the successful cultivation of the fusiform bacillus. Since then, similar claims have been published by Tunnicliff (9) Viellon, and Zuber (10),

[1] Work supported in part by a grant from the Research Commission of the American Dental Association.

FIG. 1. Stem of *Leptothrix racemosa*, showing relation of spores to central stem (arrows). Iron-hematoxylin stain.

FIG. 2. Bouquet of *Leptothrix racemosa*. Habitus view. Iron-hematoxylin stain

Ellermann (11), Muhlens (12) and others. Examination of illustrations generally accepted as evidence of a successful culture display a profound lack of similarity between the cultured forms and those found in the natural habitat. This criticism is made with the knowledge that variations may follow the transfer of organisms from the natural to the artificial environment. A careful study of the external form, genesis, pleomorphism, metachromism, and motility of the

FIG. 3. *Leptothrix falciformis*, showing stem. Fuchsin stain

fusiform bacillus will convince the careful student that these characteristics have been disregarded in comparisons made for purposes of identification. Moreover, the absence of precise descriptions makes the results of comparisons dubious.

When considering the external form, it must be realized that the fusiform bacillus is a relatively large sturdy organism, not comparable to the thin, filamentous forms found in the cultures. In shape, it ranges from a curved rod (*fig. 5*) through the falsiform (*fig. 6*), the

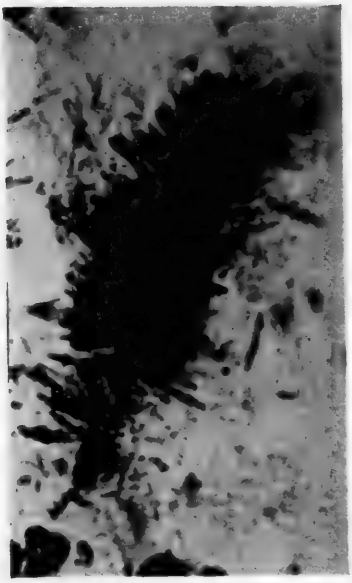

FIG. 4. *Leptothrix falciformis*, showing stem. Hematoxylin stain

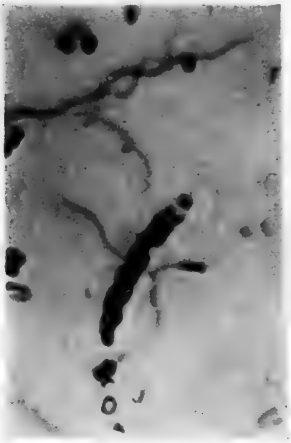

FIG. 5. *Bacillus fusiformis*, curved form. Alcoholic fuchsin stain

FIG. 6. *Bacillus fusiformis*, falciform type. Hematoxylin stain

FIG. 7. *Bacillus fusiformis*, yataghan (double curved) type. Fuchsin stain

yatagahn (Plaut) (*fig. 7*) and the straight fusiform type (*fig. 8*). The fusiform organisms occur in length ranging from a few microns to fifty microns in length. The threads which have been considered by

the author (3 and 7) belong to the heaviest type found in the mouth. The occurrence of a process of sporulation classes this organism with the algae-like fungi. The so-called metachromism of the fusiform organisms as seen by common staining methods is not a true metachromism. Through the process of drying or the use of alcohol.

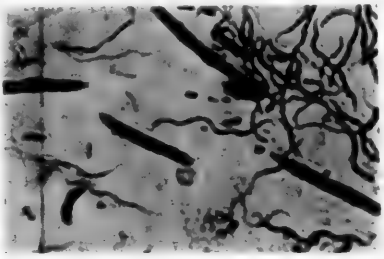

FIG. 8. *Bacillus fusiformis*, straight type. Fuchsin stain

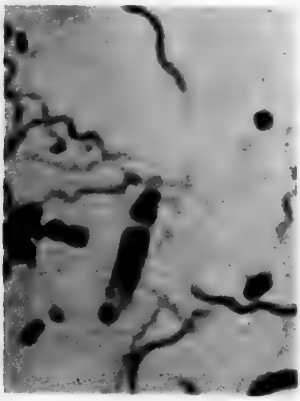

FIG. 9. *Bacillus fusiformis*, plasmolysis. Strong alcoholic fuchsin stain

organic acids or other hyperosmotic solutions employed in staining, a plasmolysis (in the sense used by Fischer (13)) occurs in these lush cells (*fig. 9*). This causes their variegated appearance. Oval bodies generally distributed with regularity and colorless or nearly so, are frequently seen within the cytoplasm of organisms stained with

Giemsa, fuchsin, methylene blue or haematoxylin. These enclosures may be vacuoles but more probably are composed of fat. One could also think of sporogenous bodies inaugurating the sporogenesis pictured above.

The fusiform organisms are motile during a part of their life. In the hanging drop preparations, a quivering of the organisms attended by an unmistakable movement across the field can be observed. They are peritrichally flagellated (3). There can be no doubt about the occurrence of these flagella. The fact that they have not appeared in cultures is a further indication that the Plaut-Vincent organism

FIG. 10. *Bacillus fusiformis*, containing oval bodies. See also *figs. 7 and 8*

has not yet been cultivated. A great variety of fusiformed organisms have been grown artificially. However, the burden of proof that one of these is identical with the organisms associated with Vincent's infection lies with the author reporting it.

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INCIPIENT DENTAL TUMOR INVOLVING PULP AND PARODONTIUM

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Tumors of the teeth and jaw are of unusual interest because they commonly develop to a more or less advanced degree before giving rise to symptoms which make their presence suspected. This insidious onset makes it impossible in most cases to visualize their exact mode of growth. The following accidentally discovered case affords an exception, since there was a complete absence of visible anatomical defects. The histology of this tumor is quite unlike any we were able to find in the literature.

In the preparation of histological material of the mouth, we desired to add to our collection a vertical section through a tooth, alveolus and mandible of a well-nourished male, mongrel dog, selected because of the apparently healthy condition of the gums and teeth. The mandible was divided by frontal section, ventral and dorsal to the first molar tooth, fixed in Zenker's fluid and decalcified in 5% nitric acid. After decalcification, the block of tissue was cut in two to facilitate subsequent dehydration and infiltration with celloidin. At this time a small, compact, grayish area, about $1\frac{1}{2} \times 3\frac{1}{2}$ mm., sharply contrasted from the rest of the pulp tissue, was observed. On closer inspection a similar mass was found in the mandibular canal adjacent to the inferior dental nerve. Examination of the stained sections revealed masses of intensely staining heterogenous material located in the following places: (a) in the pulp cavity, (b) in the mandibular canal, (c) intraperiosteally on the median surface of the mandible, (d) subperiosteally and extraperiosteally on the lateral surface of the mandible, (e) very small scattered subgingival fragments laterally and medially; also, minute fragments in the marrow spaces of the mandible and the periodontal membrane (*figs. 1 and 2*).

HISTOLOGY

Pulp—The presence of the growing mass in the pulp chamber has brought about such changes as one would expect in response to nontoxic irritation, i.e. pressure. The odontoblasts lining the pulp chamber and root canals have increased in number; in the upper portion of the pulp chamber, they have lost their characteristic shape, simulating

a reticulum of stellate cells, highly acidophilic. Approaching the tumor, the pulp tissue becomes distinctly hyperplastic, the characteristic stellate cells of the pulp are replaced by ordinary fibroblasts which assume a lamellated arrangement in relation to the tumor mass. Although this connective tissue sheath is unequal in thickness and completeness of investure, it may nevertheless represent the beginning formation of a capsule. The tumor is non-vascular, all visible blood vessels of the pulp having been crowded away from the mass. A number of small cysts are present, located principally near the pe-

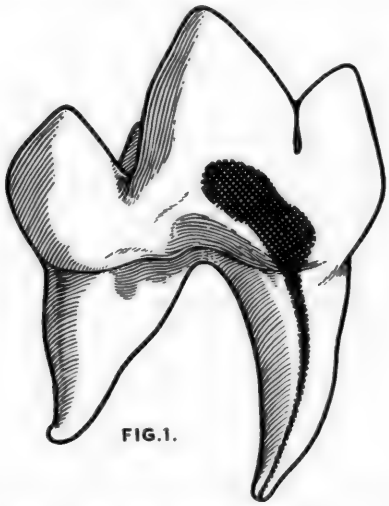

Fig. 1. Right first molar tooth of dog showing extent of tumor in pulp, as determined by serial sections.

riphery of the tumor mass, the largest at each end of the longitudinal axis. Most of these possess a thick non-cellular capsule. Only one of the larger cysts, located in the upper part of the pulp tumor, shows a slight resemblance to an ameloblastic cyst being partly filled with debris originating from stellate cells. In a few localities, deeply staining epithelial cells are seen in close proximity to these cysts, while elsewhere, elongated acidophilic cells, identified as odontoblasts, are in contact with other cysts.

The tumor mass proper consists partly of irregularly placed pieces

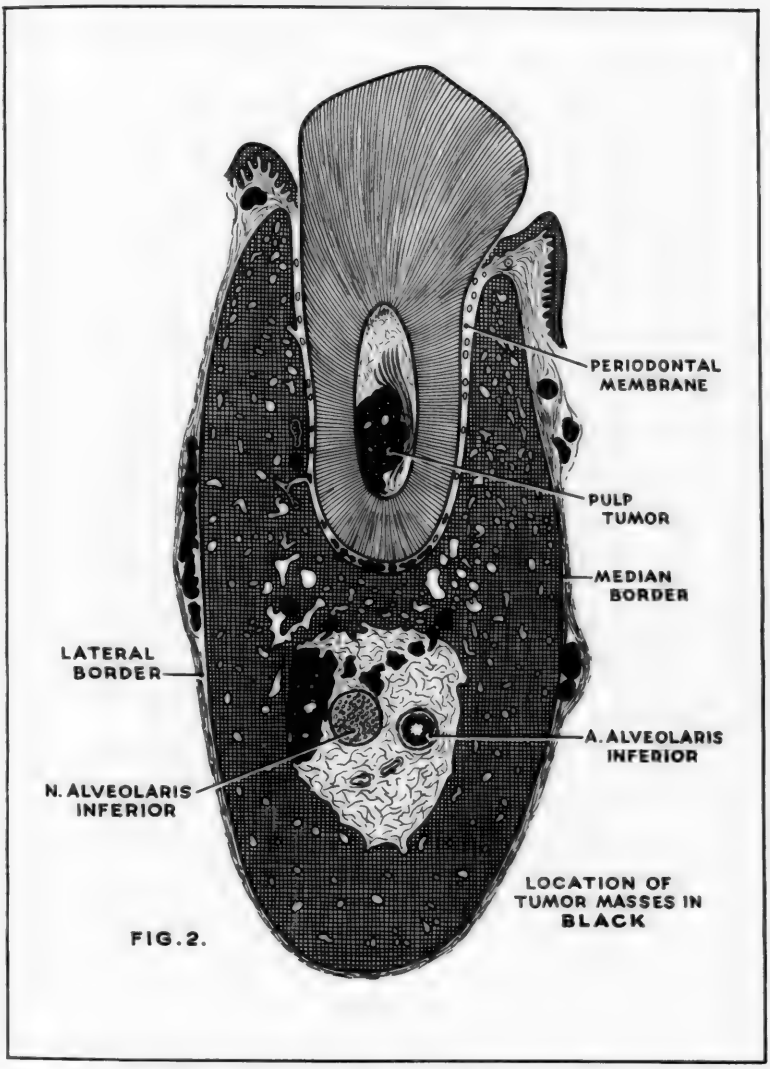

Fig. 2. Frontal section of mandible with tooth in situ (root cut obliquely). Note especially masses in apical portion of pericementum.

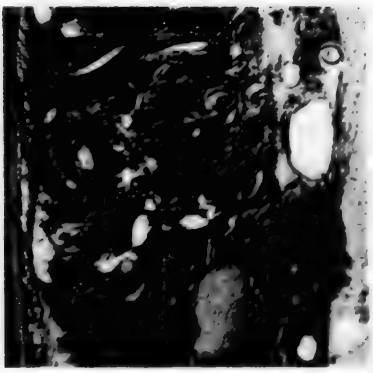

Fig. 3

Fig. 4

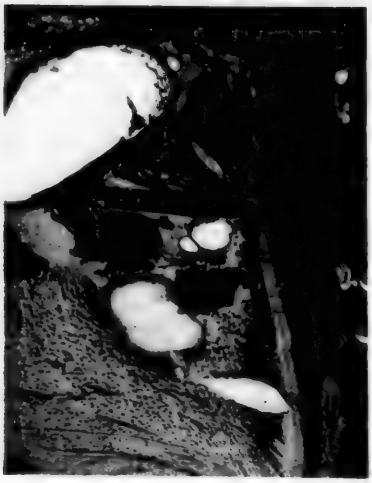

Fig. 5

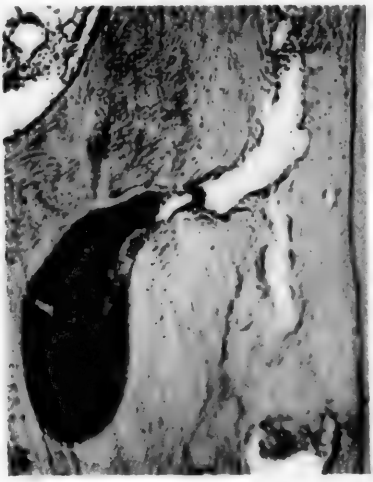

Fig. 6

Fig. 3. Tumor in upper part of root canal. Normal dentine along left border. Hairs in longitudinal and cross sections; cysts and spicules of dentine. (Orig. mag. × 60)

Fig. 4. Tumor in mandibular canal. Right border, osseous substance of mandible; tumor mass with fragments in upper left; center, inferior alveolar nerve. (Orig. mag. × 30)

Fig. 5. Detail of dentinal plate, cysts and hairs in uppermost portion of pulp tumor. One hair, longitudinal section, at left of dentinal plate. Note sharp line of demarcation between predentine and dentine, also dentinal canals. (Orig. mag. × 200)

Fig. 6. Tumor in marrow space extending from pericementum. Beginning hyperplasia of marrow in upper left hand corner. Cementum and dentine at bottom. (Orig. mag. × 200)

of dentine, with accompanying odontoblasts, the former staining a bright pink. The largest piece of dentine was 0.6 mm. in length by 0.06 mm. in thickness, cut vertically, showing a typical stratum of predentine (15 microns thick) and dentinal canals passing through the entire thickness (*fig. 5*). This plate seems to have attained its greatest development because the odontoblasts appear to be receding from it. The distal dentinal surface ends abruptly toward the adjacent heterogenous tissue mass. The odontoblasts along the plate are stratified and have greatly elongated processes which are somewhat retracted from the predentine. A number of small cysts are found between and adjacent to odontoblasts. In another location, a small piece of dentine is cut at right angles to the dentinal canals, affording an exceptionally good view of the fibers of Tome. Most of the smaller fragments of dentine are less regular in structure and shape than the above; in fact, some of them might readily be mistaken for bone. Frequently the identity of extremely irregular dentine can only be ascertained by following the spicules projecting from more typical pieces of dentine and tracing them among the other tumor debris in the form of delicate hyalinized strands. The blocks and plates of dentine are separated by a darkly staining heterogenous mass consisting partly of basophilic cells, reminiscent of prickle cells (not pigmented) and of slightly acidophilic cells containing brown pigment. The former cells show no tendency to form structures while the latter occur either as segregated cells, epithelial pearls, or in the form of follicles.

Many short hairs, not more than half a millimeter in length and from 5 to 75 microns in diameter, are found scattered throughout the tumor mass. They present a very striking appearance with their more or less clear yellow cuticula and bright red medulla with occasional pigment granules. The origin of these hairs is difficult to see on account of the advanced degeneration present. Occasionally an epithelial pearl may be encountered in the periphery of the tumor mass. Their concentrically arranged, large central cells contain many granules, while those around the periphery become more crescentic and lamellated.

Pericementum.—Many small tumors are located in the pericementum near the apical foramen. These small tumors, with few excep-

Fig. 7

Fig. 8

Fig. 9

Fig. 10

Fig. 7. Tumor extending from mandibular canal into adjacent marrow space. Metaplasia may be observed in various stages in marrow spaces above. Large marrow space to left of central dark mass contains two hair follicles and osteoid. (Orig. mag. × 200)

Fig. 8. Two adjacent marrow spaces, hair follicle in lower one, further stages of metaplasia. (Orig. mag. × 200)

Fig. 9. Marrow space in mandible, lateral to root, showing many osteoclasts. Note advanced metaplasia in marrow spaces at lower right. (Orig. mag. × 250)

Fig. 10. Earliest effect of metaplasia on osteoblasts which line up at right angles to osteal surface and eventually resemble a fibrous capsule. (Orig. mag. × 250)

tions, showed no more cellular differentiation. The entire mass, with the exception of a little of the osteoid material, is in an advanced stage of degeneration. Contrary to the appearance elsewhere, these masses were definitely surrounded by a capsule of dense fibrous tissue. In one or two localities, where the tumor extended into marrow spaces of the mandible, the capsule was missing and cellular debris in advanced stages of degeneration may be seen. Only one of the roots had these tumor masses in the apical pericementum.

Alveolar Canal Tumor—The alveolar canal tumor is located laterally to the inferior alveolar nerve. On casual examination it appears to be composed of elements similar to those in the pulp tumor. The bright red masses, however, which in the former were definitely identified as dentine, bear a closer resemblance to bone, and, therefore, will be referred to as osteoid. Furthermore, the tumor is not as compact as that previously described, being loosely embedded in the tissues of the mandibular canal. The peripheral portion of this tumor is not compact, nor is there anything present which resembles a capsule. Many sections show the presence of but a few hairs, while in other places a great many hairs are aggregated. The longest measured 1.1 mm. in length, with a diameter of 55 microns. Occasional capillaries come in contact with the peripheral portion of the mass. In a few places the tumor is fragmented, the smaller pieces extending out into the marrow spaces. The alveolar nerve and artery remain entirely free.

Marrow Spaces—The mandible was permeated with a series of small fragments of similar structure extending through continuous marrow spaces and traceable through the serial sections. The only places where the hair follicles attain a recognizable degree of development is in the larger marrow spaces in proximity to the mandibular canal. In one of these there is present a group of cells arranged in a manner strongly suggestive of a sebaceous gland (*figs. 7 and 8*). In many of these marrow spaces, one is able to observe the extension of this tumor through the gradual transformation of the structures normally present. In some of them there is a gradual hyperplasia of the supporting tissue, in others, osteoblasts form several layers, while the tissues, which these osteoblasts surround, undergo various degrees of metaplasia. Here and there where metaplasia is well under way,

remnants of epithelial cells, hairs, and osteoid material completely fill the marrow spaces. An interesting transformation is visible in the neighborhood of some of the marrow spaces which were blocked with the tumor. The osteoblasts have disappeared entirely from these spaces, and are replaced by osteoclasts arranged in regular order along the periphery. Underneath the osteoclasts Howship's lacunae are in evidence; however, the osteoclasts are separated from these a variable distance by stellate stroma cells. In the neighborhood of the larger blood vessels, one may observe isolated epithelioid cells in various stages of transformation. Thus it is possible to find normal marrow spaces and others that show various degrees of transformation to those completely obliterated by the tumor mass.

The extra-mandibular and subgingival portions of the tumor show less tendency to differentiation than the portions described before. All the tissues of the tumor are in stages of advanced degeneration, with the exception of the hairs which have apparently survived.

DISCUSSION

From the histological evidence presented in this case, we are obviously dealing with a growth originating from a misplaced embryonal remnant, dating back to a very early period, perhaps to the time when the labiogingival (labiodental) strand invades the subepithelial connective tissue. With the formation of the dento-gingival lamina and subsequently the enamel organ, some of these primitive cell remnants became detached and scattered. When the tooth finally reached adult form, some of these epithelial remnants may have remained in the pulp, while others were displaced to the parodontium (Landsberger[1]), principally the root sheath. According to many investigators, it is possible for these cell remnants to remain dormant throughout the life of an individual. Now and then some factors come into play that provoke these cells to activity. In all likelihood the provocative agent is an irritant which may be chemical or mechanical, bacterial invasions constituting agents of the former group. Serres[2] was perhaps the first to note these cell nests in the root sheath; however, he mistook them for glands connected with the teeth. It remained for Malassez[3,4] to designate these cell nests as *debris epitheliaux paradentaires* and to give them their proper significance.

With the increase in the size of this foreign cell mass, the surrounding tissues are subjected to their influence, which may be specific or non-specific, depending upon the nature of the cell remnants, the degree of degeneration of these remnants, and the potencies of the tissues with which they make contact. It is, perhaps, best illustrated in the pulp tumor. This was recognized by Tomes[5] who called attention to local thickening of the dentine, which he viewed as a compensatory change; in fact, he recorded a case of complete obliteration of the pulp cavity by new dentine, and his observation has been confirmed repeatedly by other investigators. Magitot[6] noticed that portions of teeth destroyed by caries showed new growth of protective or compensatory dentine. Gysi[7] described the exact manner of the mechanism of this dentine formation.

In the discussion of the pulp tumor, we called attention to the presence of well-formed spicules of dentine, as well as irregular masses of dentine. The small masses of dentine present in the pulp tumor, especially the plate of dentine in *fig. 5*, show quite clearly that the dentine was laid down in the form of a protective barrier against the proggress of the foreign cells. In more recent years, the question of the formation of denticles has been revived, and we find a divergence of opinions. There are investigators who believe it possible that dentine may develop free in the pulp without the presence of an irritant and through the gradual transformation of the pulp cells into odontoblasts (Weil[8], Hattyasy[9]), while others are of the opinion that denticles with the character of dentine are never found free in the pulp, but always attached somewhere to the wall of the pulp chamber (Sigmund-Weber[10], Hill[11]). They emphasize that "the apparently free denticles should be regarded as occurring in response to some irritation." Münch[12] believes that "most of the highly-organized denticles arise through an infolding process of the odontoblasts during development." He admits, however, that "odontoblasts may become separated, remaining latent, and forming denticles later." He argues definitely against osteoblast formation from the undifferentiated pulp cells.

We are unable to decide whether or not the odontoblasts have arisen from the reticulum cells of the pulp. In many places odontoblasts forming spicules are in continuity with odontoblasts lining the wall of

the chamber, while elsewhere it is impossible to trace any connection whatsoever to the normal layer of odontoblasts; in fact, were it not for the presence of the former in this tumor, we might have assumed that all of the odontoblasts had arisen through the transformation of pulp cells. It is conceivable that in the presence of proper stimuli pulp cells may transform into odontoblasts, because the odontoblasts and the reticular cells of the pulp originate from the same mesenchymal cells, the reticular cells merely remaining in a lower plane of differentiation. The occurrence of fully formed dentine and irregular dentine is, perhaps, best explained on the basis of its rapidity of growth. Römer[13], recognizing accessory dentine formation as a defense mechanism, mentions that "the quicker the pulp is called upon to form secondary dentine, the more irregular will be its formation, so the calcification does not follow regular rules and the dentine is not laid down in regular layers, but the pulp cells are included in rapidly formed irregular dentine structures." This irregular dentine has been referred to as osteoid and osteo-dentine. Even in the extradental tumor masses, the character of this material resembles dentine more closely than bone.

The basophilic cellular remnants were very difficult to identify. Some of the cells were definitely spindle shaped, while others simulated prickle cells. The addition of numerous small cysts complicated the picture. Karsner[14], in speaking of adamantinomas, calls attention to "cell nests and masses, the marginal cells of which resemble ameloblasts. . . . . These differentiate centrally either to spindle or stellate forms as to form intermediate prickle cells and keratinized epithelium." In this tumor all the cells mentioned by Karsner were present, with the addition of primitive hair follicles and hairs. While this is undoubtedly quite a rare occurrence, one must consider it within the range of possibilities, since it is the identical embryonal region which furnishes epithelium, having the potencies of forming hairs, sweat glands, sebaceous glands, the buccal salivary glands, as well as the epithelium covering the gums and forming the enamel of the teeth. The cysts mentioned principally in connection with the pulp tumor were of diverse origin. Some of them were merely cysts within massed odontoblasts, while others were partly lined or filled with

epithelial cells and remnants. A few were found without cell walls, embedded in a hyaline matrix.

The two principal tumor masses, i.e., those in the pulp chamber and in the mandibular canal, have apparently ceased to grow; also, the numerous scattered masses in the root sheath show no evidence of activity. Many of the marrow spaces, however, show interesting phases in the progression of this tumor. It is possible to see slight changes in the marrow stroma, apparently caused by mild circulatory disturbances. Frequently this is followed by the appearance of numerous osteoclasts (*fig. 9*), and the destruction of bone, perhaps for the purpose of increasing the circulation. Marrow spaces containing osteoclasts were only found near larger marrow spaces which were obliterated by the tumor mass. With the filling of a marrow space, all cellular activity as far as that particular portion of the tumor is concerned apparently has come to an end. It is impossible to distinguish anything but degenerated cell remnants, hairs, and pieces of osteoid. Rywkind[15] described an odontoma in the lower jaw of an 8-year-old child which bears some resemblance to this tumor, but it contained neither hair nor sebaceous cells.

Relative to the manner of extension of this tumor, the question arises as to its primary location. Römer[13] speaks of over-energetic pulp cells, saying that "they may here and there extend with the granulation tissue beyond the pulp chamber itself, etc." Latham[16] brings up the question of extension into the pulp from periodontal tumors. Unfortunately, the sections through the apical foramen were among the first to be cut, before the significance of the tumor was discovered. However, broken sections still on hand show definitely that the tumor completely filled part of the pulp cavity, one root canal, and its apical foramen. Apparently the circulation and nerve supply to the pulp cavity were maintained through the posterior root only. It appears, therefore, that the masses in the root sheath near the apex represent the original site of this disturbance, and that from here the cell remnants have extended both into the root canal and through the marrow spaces into the mandibular canal and out to the very surface of the mandible. This tumor was evidently a slow-growing affair, more or less self-limiting. Wherever it progressed it

interfered with the circulation, and these circulatory changes, together with the slightest variations in normal growth pressure or both, are known to be capable of inducing tissue changes which not only affect the cells normally present, but especially undifferentiated cells that might be on hand. Such cell reactions in response to histophysical alterations are discussed fully by R. Thoma[17] and Krompecher[18].

The classification of the tumor has not been stressed. The presence of hair in the tumor mass no doubt gives rise to the temptation to call it a dermoid, or the presence of the heterogenous material a teratoma (K. Thoma[19]); however, in the light of the more recent concepts of teratomas, this is out of the question (MacCallum[20]). Therefore, it is considered sufficient to call it a mixed tumor of the dental pulp and parodontium arising from the earliest period of tooth formation, in the sense of Broca's[21] classification.

SUMMARY

(1) This appears to be the first tumor on record in which hairs and hair follicles were observed in the pulp cavity in addition to the usual constituents of odontomas. (2) The presence of many early stages of the tumor in the marrow spaces of the mandible permitted observation of practically every phase of the metaplastic processes involved. (3) The centrifugal arrangement of the tumor masses in relation to the apical portion of the periodontal membrane is highly suggestive that the tumor originated from the latter.

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THE INCIDENCE OF UNILATERAL DENTAL CARIES IN 516 CASES

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Caries sometimes attacks the proximal surfaces of one tooth while the closely contacting neighbor is unaffected; this condition is defined as unilateral dental caries. An examination of 516 full mouth x-rays, selected at random, showed 179 such lesions, indicating that the condition is not rare. The results are shown graphically (*fig. 22*). Caries affecting the proximal surfaces usually attacks both teeth and the occasional occurrence of unilateral caries is not wholly explainable on the basis of the causes now ascribed to dental caries. It is believed by many investigators that the sole factors of dental caries are food fermentation, bacteria, and possibly some obscure changes in the chemical constituents of the saliva. A little thought will show that these cannot explain, in all cases, the occurrence of unilateral caries. If food is wedged between two teeth, why should not *both* of these teeth always be attacked?

An analysis of the varied probable causes of unilateral caries shows that they fall naturally into two main groups, each having a number of sub-headings: A. *Variation in Environment*—1) Bacterial plaque attached to the affected tooth only; 2) Retention of a carious deciduous tooth, causing the permanent tooth to be affected; 3) Protection afforded one tooth by an orthodontic band. B. *Variations in Resistance of Individual Teeth*—1) Calcification; 2) Structure. The beneficial or detrimental effect of the saliva can be ruled out as a determining factor in unilateral caries, because this secretion naturally bathes both closely adjoining teeth equally. The above mentioned

[1] The junior author made the preliminary examination of the full mouth x-rays and prepared the graph.

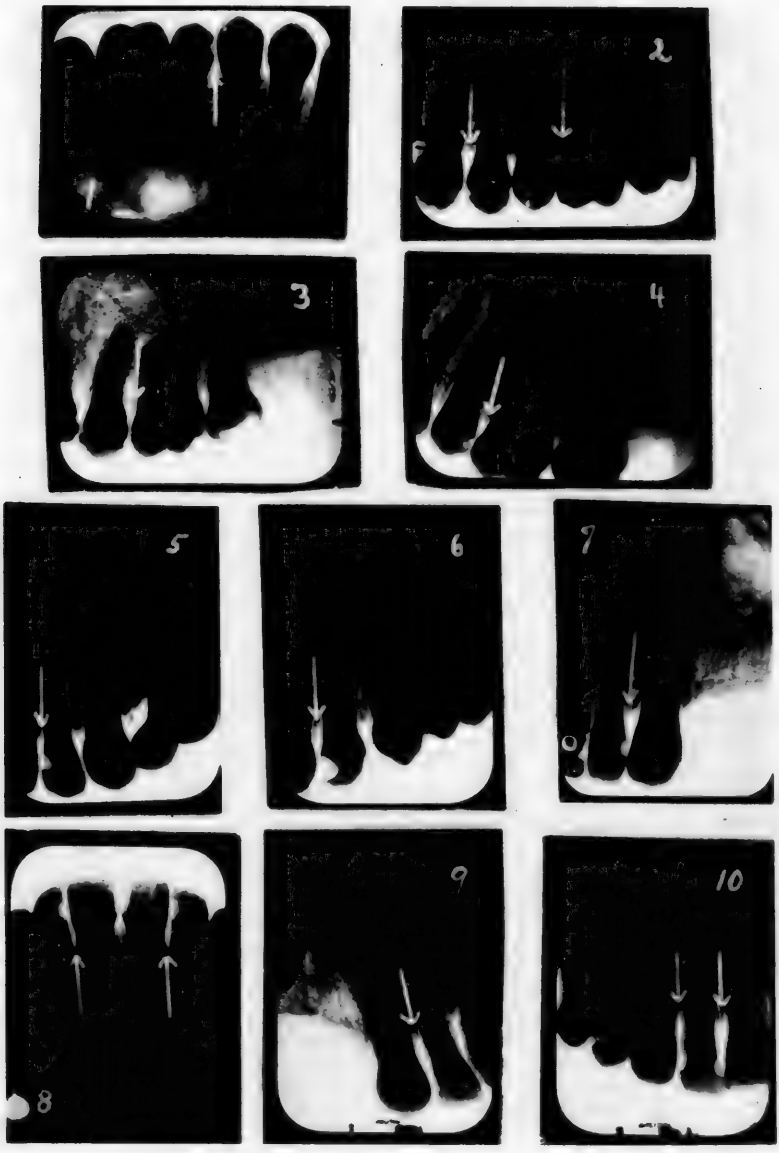
conditions which might result in the decay of only one of two closely approximating teeth are analyzed further.

Variation in Environment. 1. The presence of a bacterial plaque on the affected tooth while the adjoining surface of its neighbor remains clean seems to be one explanation for unilateral caries. Hirschfeld (1) suggested that the passage of food between two teeth may keep the surface of one tooth clean, the other being covered with food débris. While this factor may have been active in the case shown in *fig. 1*, only a few cases of unilateral caries can be explained in this manner. Some of the x-rays (*figs. 1–20*) show that the contact of the two teeth is so close that the excursion of food is impossible, thus ruling out the factor of unilateral plaques as applying to many cases of one-sided proximal caries. 2. The retention of a carious deciduous tooth may cause the adjoining permanent tooth to be affected, a condition often found in the region of the first permanent molar. The distal surface of the second deciduous molar which remains next to the permanent molar for about five years often becomes carious, permitting food retention and possibly resulting in caries attacking the mesial surface of the permanent molar before the second premolar erupts. *Fig. 2* (dotted arrow) shows such a condition in which the premolar is non-carious, and the molar carious. Owing to the fact that this is a very common occurrence at the mesial surface of the first permanent molar and that it is not an anomaly, that tooth has been excluded from the total count of the 179 lesions of unilateral dental caries. 3. Orthodontic bands may explain some instances of unilateral dental caries. When bands are well cemented, such teeth do not decay, though adjoining teeth, not being protected by bands are liable to attack.

The above observations show that certain cases of unilateral dental caries can be explained on the basis of some change in the environment of a single tooth. Many cases illustrated, however, indicate that these explanations do not apply. For instance, *figs. 3 and 4* invert the condition seen in *fig. 2*; here the distal surface of the premolar is carious, while the mesial surface of the first molar shows no such lesion. None of the above explanations apply to this condition. Twenty cases were chosen to serve as examples of unilateral caries affecting the different teeth. The observations in many of the cases

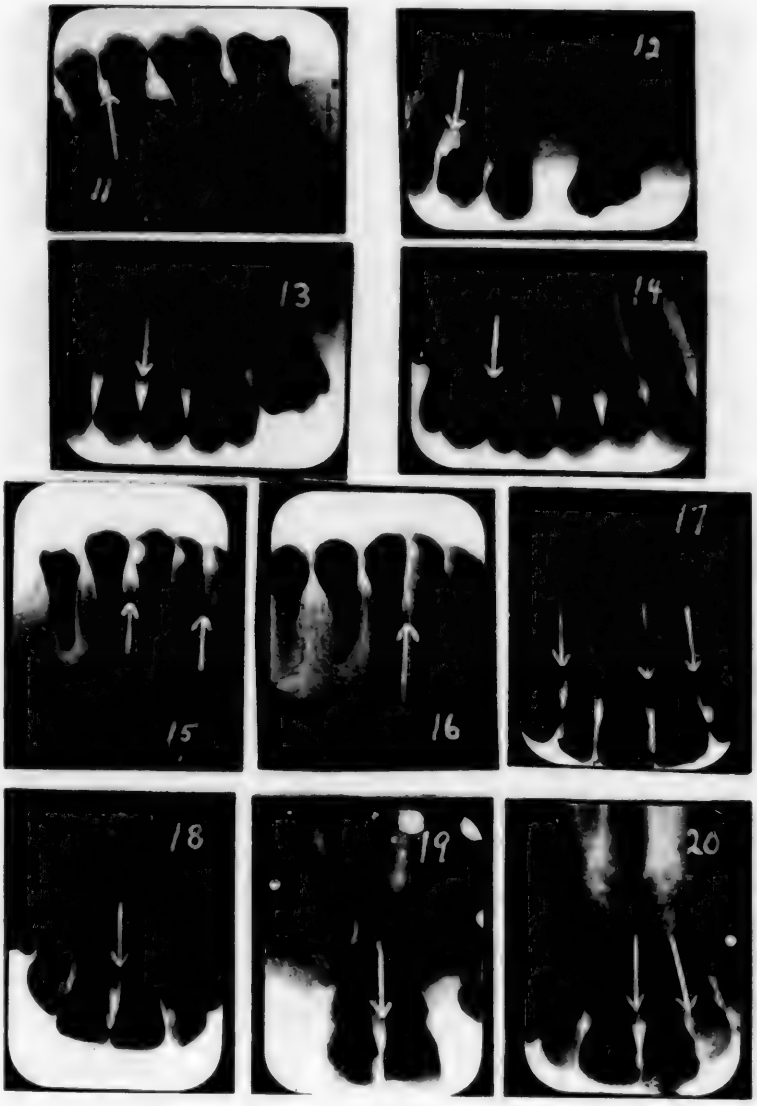
of unilateral caries throw further doubt upon the chemico-parasitic theory which regards the environmental conditions (food débris, bacteria, saliva) as being the *sole* factors in the destruction of the teeth; we will therefore pass to other possible explanations of unilateral proximal caries.

Variations in Resistance of the Teeth. 1. Insufficient calcification of a tooth may make it less resistant to caries. Black (2) claimed that there was no relation between hardness of the teeth of persons who suffer from caries and those who do not. However, with modern methods, enamel is found to vary in hardness as shown by Hodge and McKay (3) and also by Karlström (4). Similar variations in density of enamel were shown by Hollander et al. (5) using soft x-rays. The time of calcification of the crowns of the molar teeth extends over a critical period of three years immediately after birth during which time the child may go through various illnesses. Thus, these teeth may be poorly calcified, for it has been shown that a disturbance of the vitamin D-calcium—phosphorus metabolism has affected the teeth and bones of children (6). On the other hand, if such a disturbance occurs during the formation of the premolars, while normal systemic conditions prevailed during the calcification of the first molars, the former teeth would be poorly calcified, while the latter would be well calcified. Such a condition may explain the decay of the "weak" premolar (*figs. 3 and 4*) leaving the first molar intact. The varying degree of calcification may therefore be an explanation when unilateral caries affects teeth whose times of development do not coincide. Such an explanation definitely does not apply in the cases shown in *figs. 17 to 20*. A pair of first incisors is calcified at the same time, hence systemic disturbances cannot be made responsible, both teeth being developed under identical systemic conditions. Hence another explanation is offered: 2. All too little is known about the difference in structure in caries-immune and caries-susceptible teeth. Fish (7) pointed out that the presence of enamel lamellæ predisposes the teeth to caries. These structures resemble cracks in the enamel and were first described and named by the author (8). Some of them, in fact, may be cracks caused by trauma which have filled in with organic material. Such structures, probably being more permeable than the surrounding tissue, can well be, under certain circumstances,



Figs. 1–10

Figs. 1–20—X-rays of twenty-six typical unilateral lesions of dental caries (active or filled). Cavities on mesial surfaces of first molars have been excluded from study because their decay is explained by retention of carious second molar. Films selected from 179 lesions of unilateral dental caries found in examination of 516 full mouth x-rays. Fact that only one of two contiguous teeth can be affected by caries, leaving its neighbor intact, suggests that they have varying resistance to decay.



Figs. 11–20

an entrance point for the external attacking force of dental caries. One of these cases is shown in *fig. 21.* Enamel lamellæ may therefore be one explanation of the occurrence of unilateral proximal dental caries.

Susceptibility of the Second Incisor to Caries: These teeth seem to be particularly prone to proximal caries, leaving the adjoining distal surface of the central and mesial aspects of the canine intact. *Fig. 22* shows that the second incisors, particularly the maxillary, are

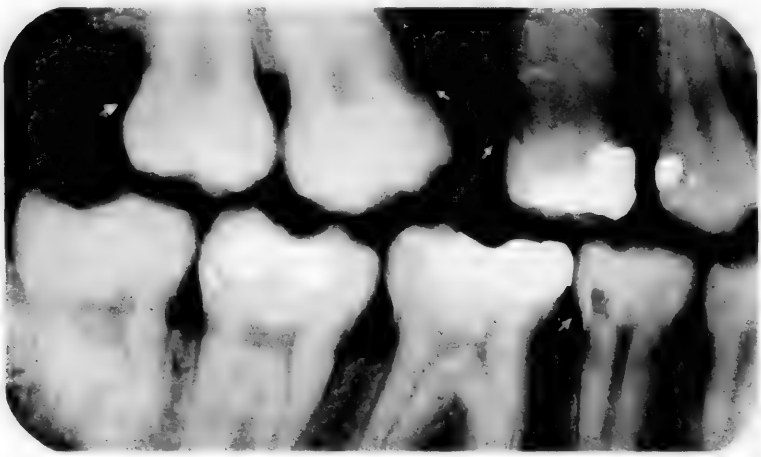


Fig. 21—Exceptionally clear x-ray showing active caries on distal surface of upper and lower premolars. Entrances to cavities (arrows) appear as dark lines penetrating from surfaces of each tooth to carious dentin; picture suggests the presence of enamel lamellae in these teeth. (Courtesy of the Eastman Kodak Co.)

affected in a greater number of cases than any other tooth in the mouth. This observation indicates that, in all probability, their caries resistance is lower than that of the first incisors and the canines. Whether a deficient calcification or structural fault make them more prone to caries is as yet unknown.

SUMMARY

An examination of 516 full mouth x-rays, selected at random, showed 179 carious lesions affecting only one of two closely contacting teeth. This condition (unilateral dental caries) indicates that food

retention, bacteria, and saliva cannot be the *sole* factors in dental destruction. A few such cases can be explained on the basis of a change in the local environment; the majority indicates that the teeth themselves have a variable resistance to attacks. The maxil-

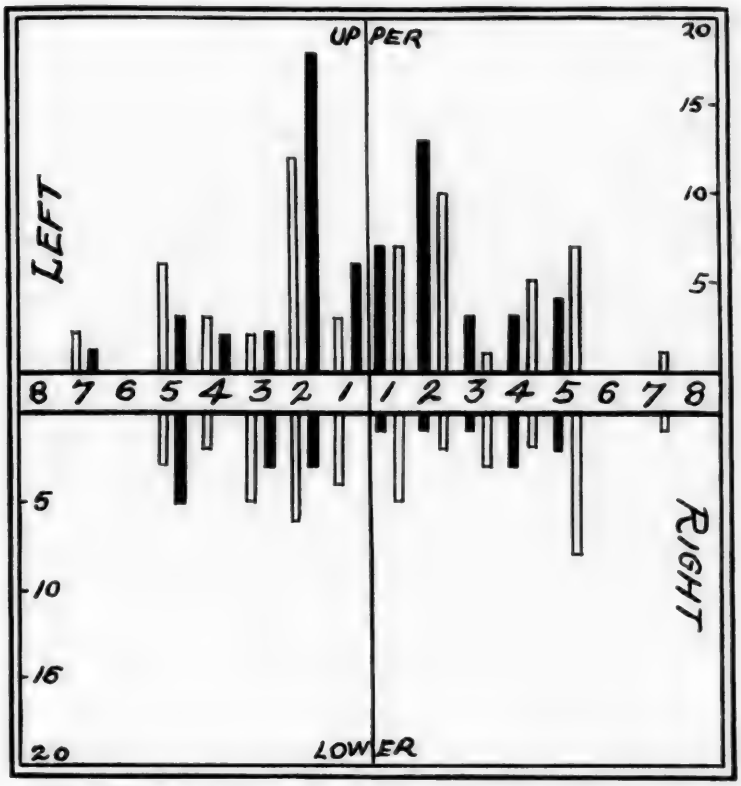


Fig. 22—179 lesions of unilateral dental caries in 103 persons observed in examination of 516 full mouth x-rays, selected at random. In many cases, maxillary second incisor effected, leaving its immediate neighbors (first incisor and canine) unaffected. First molars (6) were excluded from study.

lary second incisors seem to be most prone to unilateral caries. The principal factors affecting resistance of single teeth, as far as is known at present, seem to be variations in calcification and structural differences. Therefore, the etiology of dental caries appears to

include not only food retention, bacteria, and possible changes in salivary content, but also a variation of resistance of the individual tooth.

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SOME OBSERVATIONS ON THE pH OF SALIVA[1]

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The reaction of saliva has been the object of study by numerous investigators, either for direct reasons or because of a suspected causal relationship with dental caries. More sensitive electrometric or colorimetric methods are now employed for determining salivary pH instead of the older litmus and titration methods. In this paper, our object will be to discuss some of the major observations regarding the pH of saliva and to correlate the findings of other investigators with our own. The method used by us in collecting salivary samples has already been described (8). The apparatus employed was of the potentiometric type having a saturated calomel cell and a Cullen quinhydrone electrode. The set-up is illustrated in *fig. 1*.

The pH range of saliva has been determined by many investigators and their findings together with the method used are given in Table I. The lowest value found is about pH 5.0 (Broderick, 4.9; Cassarato, 5.0; Grossman and Brickman, 5.05); the highest pH value is about 8.0 (Broderick, 8.0; Mathur, 8.0; Grossman and Brickman, 7.9). Judgment must be reserved regarding the low pH value of 4.1 obtained by Allaria (1) and the high pH value of 8.6 obtained by Allaria (1) and by Kirk, Crowell and Appleton (2). While much credit must be given these pioneers in the early use of the electrometric method for determining the pH of saliva, it is questionable whether the apparatus then used possessed the degree of accuracy obtainable today. From an examination of available data, it appears that *the pH range of saliva is between pH 5.0 and pH 8.0.*

Few comparisons of the pH of saliva during health and disease have been made. Bloomfield and Huck (3) found no constant relationship

[1] Read before the 15th General Meeting, International Association for Dental Research, March 14, 1937, at Baltimore, Md.

between the reaction of saliva and any particular disease, but observed a slightly wider range in pH among subjects in a diseased group than those in a normal group. Our data confirm the latter observation. Sharper and more frequent fluctuations in pH were also observed by us in salivas taken from a diseased group than from a healthy group. While a drop of one entire pH unit, say from pH 7.0 to pH 6.0, was rarely observed among the members of the healthy group, it occurred in almost every instance among members of the diseased group. This observation has also been made by Saunders (4) who studied the saliva of eight epileptic patients and two controls

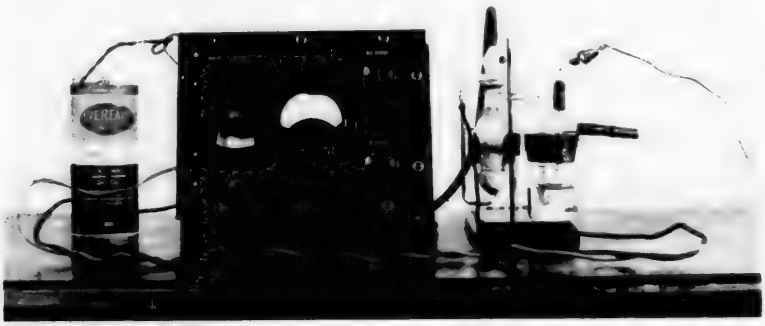

FIG. 1. Set-up of pH apparatus including saturated calomel cell and Cullen quinhydrone electrode.

every 15 minutes for from 5 to 7 hours. The controls showed only slight fluctuations in pH (about 0.5), while the epileptic patients showed much greater variation (the pH in one case dropping from 7.1 to 5.8). Also, in the epileptic group the fluctuations in pH were abrupt and marked as compared with the control cases. This suggests not only that the pH range is greater, but that fluctuations in salivary pH are more marked during ill-health. Whether this is due to emotional reactions accompanying ill-health, since Starr (16) has shown that salivary pH is influenced by emotion, or whether it is due to other factors, we are unable to say at this time. According to our data, the pH level of saliva, i.e., average pH for each individual, was

found to vary in health and disease. The pH of saliva taken from subjects in ill health was definitely lower than a normal group, the average being pH 6.4 in the former and pH 6.7 in the latter. Whether this difference is a real one or whether it is due to the fact that most of the subjects in the diseased group were at rest in bed and therefore

TABLE I

pH range of saliva

AUTHOR	LOWEST pH	HIGHEST pH	METHOD
Allaria	4.1	8.6	E
Broderick	4.9	8.0	?
Cassarato	5.0	7.0	?
Grossman and Brickman	5.05	7.9	E
Pohle and Strebinger	5.25	7.54	E
J. A. M. A.	3.25	7.86	?
Bunzell	5.35	7.41	C
Hanke	5.4	7.0	IP
Kirk, Crowell, Appleton	5.6	8.6	E
Brawley	5.6	7.6	C
MacIntosh, James, L. Barlow	5.65	7.2	IP
Starr	5.75	7.05	C
Rich	5.8	7.39	E
Bloomfield and Huck	5.8	7.5	C
Mathur	6.5	8.0	C
Englert	6.65	7.28	E
Carlson and McKinstry	6.66	7.02	C
Popper and Haffner	6.7	7.52	E
Hymanson and Davidsohn	6.8	7.8	C
Jonoff and Tschetunoff	6.8	7.6	E
Stern	6.6	7.0	C

E—Electrometric.
C—Colorimetric.
IP—Indicator Paper.

not active such as those in the normal group, we have not yet determined.

Popper and Haffner (5) investigated the constancy of the pH of saliva and found that it was fairly constant in the same subject. Our data confirm this observation, i.e., that one person may have a lower or higher pH level than another and that this level is reasonably constant for the same person. It is understood, of course, that

hourly as well as daily variations normally occur but that within certain limits, the pH of saliva is fairly constant for the same person. Three of our subjects were examined after a ten months interval. On both occasions, the average daily, average nightly, and average total pH values were either identical or showed only a slight variation of about 0.1 pH. (Table II). When it is considered that each of these three subjects had a different pH level, i.e., an average pH of 7.0, 6.8 and 6.5 respectively for 24 hours, the significance becomes more apparent. Expressing this somewhat differently, we might say that while there were hourly variations in pH in each case, a fairly constant level prevailed from which the deviation was not great. This level varied in different subjects, and we may be justified in making the

TABLE II

Constancy of pH level (average pH) in three subjects whose salivas were studied ten months apart

SUBJECT	AVERAGE pH DAY	AVERAGE pH NIGHT	AVERAGE pH 24 HOURS
A (#21)	7.17	6.70	6.95
A (#27)	7.11	6.66	6.96
B (#17)	6.99	6.49	6.83
B (#24)	6.92	6.50	6.78
C (#16)	6.65	6.25	6.51
C (#25)	6.63	6.22	6.50

following statement: *While the pH level of saliva varies with different people, it is fairly constant in the same person.*

The "average" pH of saliva has been determined by a number of investigators, the average being based upon different data. For example, the "average" might be based either on daily, hourly, or "from time to time" observations. Or again, the average might not take into consideration the pH of saliva during sleep. That the pH of saliva during sleep does vary from that during the day when one is active will be discussed later. The word "average" will be used here only in the sense that other authors have used it, i.e., average pH of saliva based upon determinations made during the day. Recent investigators, with few exceptions, have found the average pH of

saliva between pH 6.5 and pH 7.0. (See Table III). Our data show that the average pH of samples of saliva taken during the day from normal, healthy subjects is 6.7. This is very close to the diurnal average of pH 6.75 observed by Brawley (6) who made an extensive study of salivary pH, and is within 0.2 pH of the average given by the majority of investigators. From an examination of available data,

TABLE III

Average pH of saliva

AUTHOR	CASES	OBSERVATIONS	pH	METHOD
MacIntosh, James, Lazarus-Barlow	12		5.6–5.9	IP
Bunzell	298		6.0–6.64	C
Cassarato	100		6.35	C
Koneffke			6.4–6.6	?
Starr	228		6.6	C
Allaria	51	51	6.6	E
Bloomfield and Huck	52	102	6.6–7.1	C
Grossman and Brickman	46	505	6.7	E
Brawley	3,405		6.75	C
Stern	100		6.8	C
Rich	57	171	6.82	E
Carlson and McKinstry	127		6.85	C
Karahan, Krasnow, Krejci			6.9–7.0	C
Englert	2	112	6.98	E
Jonoff	412	412	7.0	C
Hymanson and Davidsohn	55	100	6.8–7.8	C
Mathur	31	135	7.5	C

E—Electrometric.
C—Colorimetric.
IP—Indicator Paper.

it may be concluded that *the average pH of diurnal saliva is between pH 6.5 and pH 6.9.*

So far as we have been able to determine, the only reference in the literature to a possible difference in pH between diurnal and nocturnal saliva is that of Pohle and Strebinger (7). They made pH determinations from about 6 A.M. (on arising) to 2 A.M. (on retiring). About twelve samples of saliva were examined during this time and, according to their observation, the saliva became more alkaline during the night. This is contrary to our findings. Since no data are presented by Pohle

and Strebinger and since their observations were not made on subjects during sleep, their conclusions are not altogether valid. We have shown (8) that the pH of saliva drops during the night, or more specifically, during sleep. In a controlled group of 25 persons on whom hourly observations for a 24 hour period were made, the diurnal pH was 6.7, while the average nocturnal pH was 6.4. In another group of 11 persons, where similar studies were made, the nocturnal pH was

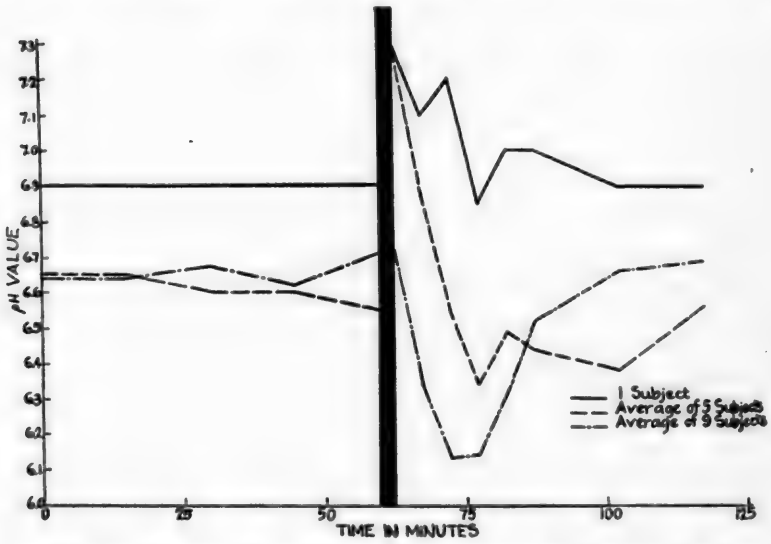

FIG. 2. Effect of eating on pH of saliva. Heavy vertical line indicates time when food was given.

also lower than the diurnal pH. Our data indicate, therefore, that *saliva has a lower pH during sleep than while awake.*

References to the effect of eating upon salivary pH have been made from time to time, but the resulting effect has been rarely mentioned. In controlled experiments, Mathur (9) found that the pH of saliva changed with the first morsel of food taken into the mouth, that there was a rise in pH during eating followed by a decided drop, and that saliva returned to its original pH value after some time. The effect of eating on the pH of saliva was also studied by us. For purposes of

control, pH determinations were made every 15 minutes for at least an hour before eating. After eating a meal, pH determinations were made every 5 minutes for 30 minutes and every 15 minutes for the remainder of the hour. Fifteen subjects were studied. In 5 cases, an immediate rise, followed by a marked drop in pH occurred before a return to the original pH level. In one case, a rise without a drop below the normal pH level occurred. In the remainder, a drop in pH, not preceded by a rise, occurred. (See *Fig. 2*). From this we may conclude that eating lowers salivary pH, and that this drop is sometimes preceded by a rise in pH. A return to the original pH level occurs within about one hour.

SUMMARY

(1) The pH range of saliva is from pH 5.0 to pH 8.0. (2) The pH range of saliva among those in ill health is greater than among those in good health. (3) Although the pH level of saliva varies with different people, it is fairly constant for the same individual. (4) The average diurnal pH of saliva lies between pH 6.5 and pH 6.9. (5) The nocturnal pH of saliva is lower than the diurnal pH. (6) There is a drop in pH of saliva after eating. This drop in pH is sometimes preceded by a rise in pH.

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FACTORS AFFECTING THE INCIDENCE OF DENTAL CARIES IN POPULATION GROUPS[1]

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The survey recently made and the results published (1) under the auspices of the U. S. Public Health Service have made available for the first time mass statistics on the comparative incidence of dental caries in population groups. While one may perhaps question the *absolute* accuracy of the data collected, the method of its collection at least gives it a considerable value for comparing one region with another. The undertaking was an enormous one, and, although the end results fell far short of those desired, the published statistics do yield valuable information.

Beyond the observations of Price and others (7) on the relative freedom from caries enjoyed by people living under primitive conditions, and the early onset of tooth decay and dental abnormalities when they become contaminated by trade or intercourse with civilized groups, little has been known of the incidence of tooth decay over the earth. It has generally been held to be less prevalent in our southern states than in the North, but definite comparative figures have been lacking. Because of its importance in the general health of individuals, tooth decay ranks as a health problem of no mean proportion, so that it becomes worth while to secure a general view of it as it varies from region to region and group to group. In the present communication will be presented summarized data of such regional and group relationships, with a discussion of their possible significance in regard to caries causation and prophylaxis.

Latitude and Caries Incidence. Using data taken from Public Health Bulletin #226 (1), an index of caries incidence was derived as follows: Boys of the 12–14 year age group were used throughout the

[1] Presented at the 15th General Meeting of International Association for Dental Research, Baltimore, March 14–15, 1937.

study, the oldest age group being used to show as much as possible of the defects in permanent teeth, and boys being used in preference to girls so as to avoid some of the metabolic disturbances of puberty which comes on earlier in girls and shows greater differences in age at onset (3) in different regions. From the data in the Bulletin, addition was made of the number of carious teeth per 100 children, both deciduous and permanent, as listed under (*a*) ***present dental needs and oral pathology***, and of deciduous and permanent teeth filled and permanent teeth extracted per 100 children, as listed under (*b*) ***past dental***

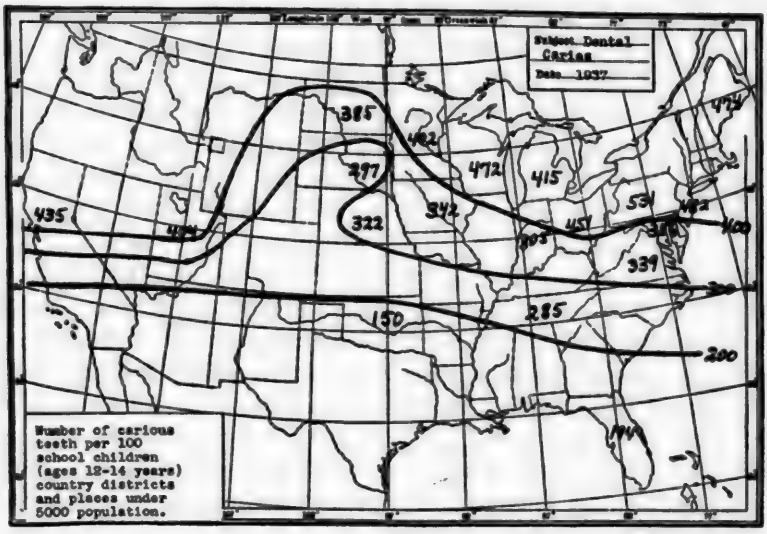

FIG. 1. Dental caries in school boys (12–14 years) of the United States

treatment. The sum of all teeth carious at the time of examination and of those already filled or extracted (permanent only) gives a satisfactory index of the progress of caries for the given age period.

In *fig. 1* are presented the indices of caries thus derived for "County balances" (school children living in rural areas or towns of less than 5000 population) in some 19 states. The numbers represent arithmetical averages of all county indices for any given state. In this map showing the gross relation of caries to latitude larger cities, which would be more likely to have treated well or river water, are

excluded and a regional comparison of caries incidence is made with populations more likely to use only well water. *Fig. 1* shows very clearly the increasing incidence of caries from south to north, the increase being roughly 15 more decayed teeth per 100 children for each added degree of latitude, or well over 200 per cent from Gulf to Canadian border. The immediate explanation that comes to mind is the decline in sunlight intensity toward the north, since the relation of ultra-violet light to bone and tooth development has already been so clearly demonstrated. Supporting this explanation is the lessened

TABLE I

Relationship between caries and latitude in cities of 10,000 or more population

NUMBER OF CITIES	LATITUDE	AVERAGE NUMBER OF CARIOUS TEETH PER 100 SCHOOL CHILDREN, AGES 12–14 YEARS
5	25–32.9°	271
4	33–34.9°	296
7	35–35.9°	299
6	36–36.9°	291
6	37–37.9°	380
5	38–38.9°	307
15	39–39.9°	447
72	40–40.9°	476
21	41–41.9°	447
12	42–42.9°	467
12	43–43.9°	527
9	44–44.9°	465
5	45–45.9°	482
2	46–46.9°	587
3	47–47.9°	337

incidence of caries in the upper Plains states as compared to other northern states where sunlight is less plentiful, especially through the winter season. Cities of 10,000 or more population display a similar general dependence of caries on latitude, although much greater individual variations are brought about by differences in hardness of drinking water from municipal systems as will be seen later. (Table I).

There have been previous indications that caries is less prevalent in warmer regions, but never before has such definite evidence been

at hand as is supplied by the data here shown. R. A. Kehoe (3) says that, during a series of physical examinations on a considerable number of Mexicans of all ages in an isolated region of central Mexico, he saw practically no caries; dental service facilities and pyorrhea were likewise non-existent. We may, I believe, accept as a demonstrated fact this low incidence of caries in tropical warmth, and a progressive increase outward toward the cooler temperature regions. Whether the increase northward continues into the polar regions cannot yet be said. Price (7) has described nearly perfect dental conditions in Eskimos untouched by civilization, although they rapidly deteriorate in contact with white civilization. In data from Bulletin 226 there is some indication of a lessening incidence of caries in northern Minnesota. In the upper half of Minnesota 18 counties supplying data show an average of 380 carious teeth per 100 children (12–14 yrs.) while in the southern half 49 counties have an average of 408. Likewise, the only two Minnesota cities above the 47th parallel included in the survey were Virginia and Hibbing with 349 and 230 carious teeth per 100 children respectively, while the state average for cities is 474. Data from northern Wisconsin and Michigan were too scanty to be of value for comparison.

Caries in Relation to Slope of River Drainage Areas. In addition to the relation of caries to latitude, there also seems to be some degree of dependence on slope of terrain; caries tending to increase as one passes from the head waters of a river toward its mouth. Where whole states have a more or less general slope, caries is less prevalent in people of the higher region. In Oklahoma, for instance, the general slope is downward to the east, and of 8 counties supplying data the 3 western ones gave an average of 109 carious teeth per 100 children while the 5 eastern ones gave 174. Of 8 Oklahoma cities for which data are available, the 4 western ones averaged 199 carious teeth per 100 children and the 4 eastern 321. In North Dakota, with a similar slope eastward, 5 western counties averaged 310 and 10 eastern counties 423 carious teeth per 100 children. No counties in western South Dakota were included, but in the eastern portion of the State with a general downward slope to the south, 12 northern counties averaged 262 carious teeth per 100 children and 11 southern counties 333. Here

the latitude effect was overshadowed by that of terrain slope. Following up the course of certain rivers, We also see a marked drop in caries incidence. With the Whitewater river of southeastern Indiana the following county averages are found, as one travels up from the river mouth to its headwaters:

Up the Whitewater River (Indiana)

Dearborn County		415
Franklin	"	382
Fayette	"	233
Wayne	"	304
Randolph	"	157

Up the Wabash River as it crosses Indiana

Vigo County		332	Cass County		292
Vermillion	County	365	Whitely	County	182
Tippecanoe	"	360	DeKalb	"	182
White	"	331	Adams	"	206
Carroll	"	327	Jay	"	104

Up the Miami River in Western Ohio

Cincinnati	817
Hamilton	397
Lima	253

Up the Ohio and Scioto Rivers

Cincinnati	817
Portsmouth	651
Columbus	396
Lima	253

There would therefore seem to be some factor in soil or water that tends to produce less caries in the upland regions or head waters of rivers and more caries down nearer the river mouth. Whether this be greater leaching of the soil in the lower regions or greater hardness of drinking water in the upland cannot yet be said.

Relationship of Drinking Water Hardness to Caries. It is obvious that calcium and magnesium supply in drinking water and food may play a considerable part in tooth and bone formation and in tooth decay. Experiments with controlled diets have amply demonstrated this to be true in animals. To see if such a relationship could be demonstrated for human population groups all the cities, for which data were given in the report (1), were requested to submit data as to the source, method of treatment, and degree of hardness of their city water as supplied to the mains. Replies from 75 cities are at hand and yield the following information:

NUMBER OF CITIES	MEAN LATITUDE	SOURCE OF WATER	CHLORINATED	AVERAGE HARDNESS	AVERAGE CARIES PER 100 CHILDREN
53	40.76°	Rivers and Lakes	All	113 p.p.M.	460
11	40.81°	Wells and Springs	None	246 "	387
11		Wells and Springs	All	285 "	325

There was found to be *almost 30 per cent more caries* among children of cities using river and lake water than among those using water from wells or springs, even though the mean latitude of the two groups was practically the same. The reason for this difference, however, may well reside in the degree of hardness, for it is seen in the data given above that caries diminishes as hardness increases. A more detailed grouping of the 75 cities regardless of water source further exemplifies this fact.

NUMBER OF CITIES	HARDNESS (PARTS PER MILLION)	AVERAGE CARIES PER 100 CHILDREN
16	Below 50	496
14	50–99	397
20	100–199	476
10	200–299	355
10	300–399	382
3	400–499	342
1	500+	318

A list of the cities used in this analysis of the relation between drinking water hardness and caries is given in Table II. Outstand-

TABLE II

Caries in relation to source and hardness of city drinking water

STATE	CITY	WATER SOURCE	HARDNESS, PARTS PER MILLION	CARIES, NO. PER 100 CHILDREN (12–14 YRS.)
Florida	Tampa	Hillsboro River	101	314
	Jacksonville	Artesian Wells	300	250
Arizona	Tucson	Deep Wells	260	177
	Phoenix	Verde River	200	349
		Sub surf. flow		
Georgia	Atlanta	Chattahoochee R.	20	312
Oklahoma	Tulsa	River	95	202
Tennessee	Memphis	Artesian Wells	30	422
	Chattanooga	Tennessee River	30	268
	Nashville	Cumberland River	90	242
	Johnson City	Mt. Springs	63	277
Colorado	Pueblo	Arkansas River	300	223
	Denver	S. Platte River	120	375
	Colorado Springs	Stream, Reservoirs	25	206
	Fort Collins	Cache LaPoudre R.	17	286
Utah	Salt Lake City	Canyon Streams	175	417
Virginia	Winchester	Springs	280	500
Indiana	Indianapolis	White River	290	492
	Peru	Wells	500	318
	Logansport	Eel River	286	431
	Elkhart	?	225	219
	Michigan City	Lake Michigan	136	451
	Gary	Lake Michigan	120	308
	Marion	Deep Wells	430	274
	South Bend	Artesian Wells	342	444
	Goshen	Deep Wells	325	409
Ohio	Norwood	Deep Wells	383	544
	Cincinnati	Ohio River	110	817
	Hamilton	Wells	118	397
	Columbus	Scioto River	87	396
	Canton	Deep Wells	340	475
	Lima	River	220	253
	Toledo	Maumee River	240	461
Pennsylvania	Pittsburgh	Allegheny River	100	529
	Easton	Delaware River and Wells		476
	Reading	Maiden Creek	80	680
	McKeesport	Monongahela River	90	376
	Johnstown	Impounded Mt. Streams	23	269
	Pottsville	Creeks and Runs	10	710

TABLE II—*Concluded*

STATE	CITY	WATER SOURCE	HARDNESS, PARTS PER MILLION	CARIES, NO. PER 100 CHILDREN (12–14 YRS.)
New Jersey	Hackensack	Hackensack River	75	321
	Englewood	Hackensack River	75	454
	Lodi	Hackensack River	75	451
	N. Bergen	Hackensack River	75	405
	Patterson	Passaic River	50	372
	Newark	River Reservoir	30	439
	Nutley	River Reservoir	30	466
	New Brunswick	Lawrence Brook	44	381
	Rahway	Rahway River	110	470
	Elizabeth	Wanaque River	30	483
	Clifton	Wanaque River	30	532
Nebraska	Hastings	Deep Wells	164	320
	Grand Island	Wells	118	348
Iowa	Davenport	Mississippi River	145	624
	Des Moines	Raccoon River	300	414
S. Dakota	Huron	Wells	224	352
	Sioux Falls	Shallow Wells	430	440
N. Dakota	Grand Forks	Red Lake River	400	433
Minnesota	St. Paul	Miss. R. Lakes and Deep Wells	165	580
	Minneapolis	Mississippi River	172	553
	Virginia	Mine drainage pit	170	349
	Hibbing	Wells and Mine pit	180	230
	Duluth	Lake Superior	54	607
Wisconsin	La Crosse	Wells	304	255
	Milwaukee	Lake Michigan	130	832
	Two Rivers	Lake Michigan	130	589
	Manitowoc	Wells	350	631
Michigan	Detroit	Detroit River	98	512
	Flint	Flint River	285	317
	Port Huron	St. Clair River	100	506
	Saginaw	Saginaw River	100	340
	Lansing	Deep Wells	390	239
Massachusetts	Fall River	Watuppa Lake	100	572
	New Bedford	Ponds	10	578
Maine	Biddeford	Saco River	17	443
	Portland	Sebago Lake	16	895
Oregon	Portland	Bull-Run River	3.5	629
	Medford	Big Butte Springs	34	432

ingly high in their caries incidence are most of the cities of the mining regions of Pennsylvania and those of the Ohio River down as far as Cincinnati. The reason for this leads to interesting speculation but needs further factual evidence before definite statements can be made. Studies on lead poisoning, and treatment to remove lead stored in bones (2), have shown that the ingestion of certain acids, such as hydrochloric, sulphuric or phosphoric, will cause liberation of calcium from bones and teeth in large amounts, and its excretion in the urine. Oxidation within mines results in the formation of large amounts of sulphuric acid which seeps out into streams and rivers in these mining regions and makes them strongly acid in reaction. Millions of tons of sulphuric acid seep into Pennsylvania streams yearly and affect Ohio River water as far down as Cincinnati before sufficient dilution takes place to cover the acidity. In addition to the corrosive and destructive action of this acid on boilers, metal pipes and river craft, it may well be the factor largely responsible for the high caries rate in the mining regions and down the Ohio River. There is, in addition, the possible effect of oxidized sulphur liberated into the air by coal combustion in homes, factories and railroad engines. Such oxides, breathed into the lungs, are probably dissolved and retained in the body where they exert an acid effect. Analysis should be made of data on caries incidence in certain smoky cities in relation to smoke contamination of city air and its possible bearing on caries incidence.

In the Cincinnati metropolitan district there is afforded a striking instance of a wide difference in water hardness being accompanied by a considerable difference in caries incidence. Norwood, a city of 30,000 population, is entirely surrounded by Cincinnati but has its own city water system, the entire supply from deep wells is pumped into the mains without treatment. Cincinnati uses Ohio River water with a total hardness, after treatment, of around 110 parts per million, as contrasted to 383 p.p.m. in the Norwood water. Norwood children were not included in the Public Health Survey, but similar data obtained on 435 school children averaging 13 years of age showed 544 carious teeth per 100 children, which is in sharp contrast to the Cin-

cinnati figure of 817. Here are two cities with contiguous populations, but with marked difference in drinking water and dental caries. Industrial use of the hard Norwood water has frequently raised problems in the prevention of boiler scale, but there would seem to be compensating advantages for its use as drinking water.

Relation of Salt Intake in Food to Caries. As was shown in the lead excretion studies previously referred to (2), halide salts of alkaline or alkaline earth metals possess, when ingested, an action similar to that of fixed acids in promoting calcium withdrawal from bones and teeth and its increased excretion through the kidneys. Others (6) had previously shown that increased NaCl ingestion in animals caused the excretion of greater quantities of calcium and phosphorus in the urine. It was, therefore, deemed advisable to investigate the possible relation of salt intake to caries incidence in man. Price (7) has remarked how primitive people so promptly begin to show dental decay and abnormalities after contact with civilization. One of the first articles of trade brought to such primitive people is salt. Salt, sugar and white flour form the staple articles of barter for whatever the natives have to trade.

There exist wide differences in home use of salt on food, even among members of the same family. A survey was conducted among the pupils of Norwood High School, Norwood, Ohio. Cards were distributed on which each pupil wrote his or her name, date of birth and answers to the following questions:

Do you add salt to your food at the time of eating?

If so, do you add little, or much?

This gave three groups, those that added no salt to their food, beyond that used in its preparation, those that added little, and those that added much. It was not deemed advisable to try for greater detail with such individuals (7th to 12th grades). Data on caries incidence were next taken from the records kept by the school dentist in his yearly examinations of all students. All teeth that had been or were at the time carious, whether filled, extracted or untreated, were counted and the total number for the individual noted on his card. The cards were then sorted by age, sex, and salt grouping, with the following findings:

Salting of food and caries incidence

AGE (BOTH SEXES)	NO SALT ADDED		LITTLE SALT		MUCH SALT	
	Aver. no. carious teeth	No. of pupils	Aver. no. carious teeth	No. of pupils	Aver. no. carious teeth	No. of pupils
13	5.00	30	5.64	107	5.40	25
14	6.69	56	6.82	109	7.22	23
15	7.04	68	7.39	166	7.80	34
16	7.73	62	7.92	140	8.85	46
17	8.72	43	8.54	102	9.51	37
GIRLS ONLY						
13	5.33	33	5.79	56	5.80	10
14	6.22	36	7.11	47	6.86	14
15	7.33	36	7.66	71	8.81	16
16	8.18	39	8.30	76	8.79	24
17	7.61	18	8.65	55	9.30	20

Total No. boys (12–19 yrs.) = 550, average caries = 7.44.
Total No. girls (12–19 yrs.) = 617, average caries = 7.42.

There is evidence in this data, either for the whole group or for the girls alone, of a trend toward more caries as salt use increases. The differences do not rise to proportions that are mathematically significant, due to the small number of cases in each group, but the trend in the group differences is quite evident. In the 16 year group for both sexes combined, the difference between the average number of carious teeth in those using no salt and those using much salt was 2.2 times its own probable error, which means that such a difference would occur by chance only once in 6.25 times. Combining all age groups, the caries rates were:

For the "no salt" group —7.114 (299 cases)
For the "little salt" group —7.406 (687 cases)
For the "much salt" group—8.017 (181 cases)

Tests have been planned to see what correlation will exist between salt content of diet and resistance to breaking in rat incisors.

One further point worth noting in the data from the salt survey is that the incidence of perfect sets of teeth was much higher among those adding no salt (1 in 27) than among those using little (1 in 45) or much salt (also 1 in 45).

DISCUSSION

This analysis of factors affecting the distribution of caries in human populations has provided general confirmation for past observations on the mineral metabolism of bones and teeth in experimental animals. The effect of ultraviolet light, for instance, is just as evident in the latitude gradient of caries in man as it is in experimental animals. Lessened hardness of drinking water or increased salt intake tend to increase tooth decay in man much as they have been shown to diminish calcium storage in animals. The suggested effect of fixed acids tending to increase caries also fits in well with the known dissolving action of such acids on bone and tooth minerals. The facts and general relationships presented would tend to support the idea that caries is basically a metabolic disturbance, and only secondarily a matter of infection. There may perhaps be some significance attached to the fact that caries in its geographic distribution in America rather closely simulates the distribution of other evidences of metabolic breakdown under the driving force of climatic stress. Valuable results might well accrue from further investigation along this line. Tooth decay is of sufficient importance as a general health problem to warrant intensive studies along any line that offers likely leads as to its basic causation or control.

The part drinking water seems to play is well worthy of consideration. If the findings here shown for human subjects can be corroborated on experimental animals, it may be advisable to recommend the use of properly constituted bottled waters for those troubled with active caries. We may even come to the point of recommending that whole population masses provide themselves with drinking water of proper constitution in areas where city water supplies are too low in hardness or otherwise deleterious. Standards for drinking water can no longer be considered only on bacteriologic grounds or freedom from contamination, if proper regard is to be given to tooth preservation. Industrial and domestic uses of water show very definite conflict in the constitution desired and even in domestic use alone there is conflict.

Should further study on experimental animals confirm the effects of salt intake noted on school children, then again will be provided a

basis for definite prophylactic advice. It is possible that a sharp curtailment of salt intake, together with the use of special drinking waters, might go far to control the cases of active caries that proceed so destructively under ordinary handling.

SUMMARY

In a general way the survey data show the following points. First, *the incidence of caries in American school children increases steadily throughout the United States as the distance from the tropics increases.* This trend is in evidence in both rural and urban populations. Its probable relation to sunlight intensity is further supported by the presence of less caries in the northern Plains states, where sunlight is more plentiful than in other northern areas. Second, *there are indications of diminishing caries incidence as one passes from the mouth of a river toward its head waters,* a phenomenon that may be related to soil leaching. Third, *there is an inverse relationship between drinking water hardness and caries incidence, cities using river water showing about 30 per cent more caries than do cities using well water.* Fourth, *there is a direct relationship between caries incidence and amount of salt used in seasoning food.* Fifth, *negro children in all areas show less caries than do the white,* but the same regional relationships obtain.

CONCLUSIONS

Tooth decay shows certain definite relationships to extrinsic factors. It is related to latitude, increasing steadily toward the north within the United States. This is probably related to the known beneficial action of ultraviolet light in promoting tooth and bone formation and repair. Caries is also related to hardness of city drinking water, and to dietary salt intake. It is suggested that the high caries incidence of mining regions may be due to the sulphuric acid seepage from the mines into the water used for drinking purposes. The possible rôle of sulphur oxides liberated in coal burning may likewise be a factor tending to increase caries, due to the absorption of these oxides in the lungs and their action as fixed acids on the mineral metabolism of the teeth.

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THE RELATION BETWEEN DIET AND CARIES IN THE SOUTH AFRICAN BANTU[1]

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In a previous communication (2) it was reported that the South African Bantu, relatively free from caries while living under primitive kraal conditions, acquired the Europeans' susceptibility to dental decay as they adopted European ways of living. It was shown that an increased incidence of caries was associated with eating (a) sugar and (b) machine ground mealie meal. The work was repeated on a different series of cases to amplify and establish, or to refute, the conclusions previously drawn. The object of this and the previous work is to discover why civilization brings decay to the Bantu teeth.

METHODS

To collect the required data the authors undertook an extended tour through the native territories of Sekukuniland, Swaziland and Zululand and visited several dozen primitive native kraals. As there were few individuals of the desired age group (15–25 years) at each kraal, as the kraals were many miles apart with poor communication, and as it was difficult to convince each kraal of the *bona fides* of the party, another plan was necessary for collecting the hundreds of cases required. The Witwatersrand Native Labour Association, which has a central compound in Johannesburg through which several hundred thousand natives pass each year on their way to work on the various mines, gave the authors the services of two interpreters and access to as many cases as they could handle. The cases chosen were healthy young males between 15 and 25 years of age. In order to avoid those who had come under too much European influence (i.e. in order to observe those in the transition stage between primitive and European ways of living) the only cases examined were those who had worked neither in the town nor the mines previously.

Within an hour or two of their arrival at the compound suitable cases were given a thorough dental examination by one of us (J.N.N.), then collected into a group and kept away from other natives until after their interrogation. The interpreter gained the confidence of each individual, examined independently, and used a standard technique of interrogation. The question "What do you eat in your kraal?" was asked and the in-

[1] Presented at a meeting of the Royal Society of Medicine, Odontological Section, London, 1936.

dividual was given a minute or two to think it over. When each had mentioned as many items as occurred to him, questions were asked regarding items not mentioned. As a rule the native had no difficulty in deciding whether he was in the habit of eating any particular article of food regularly (i.e. several times a week), occasionally, or not at all. The fact that the natives gave the information directly and without hesitation, and that they had nothing to gain by giving misleading answers, satisfied the authors that the data obtained, though obviously not exact, were reasonably reliable. As a check, in each group, a few cases were questioned independently by the other interpreter. The duplicates usually showed only one or two minor differences; major differences did not occur more than two or three times in the course of the investigation.

A short explanation of some of the foods is necessary for those not acquainted with the habits of the Bantu. *Fresh milk* is not a common article of food except among the Bechuana; among the others, apparently, those who take fresh milk are principally natives using European foods such as tea and coffee. Some of the primitives are a little shocked at the idea of eating what they call "raw" milk. *Sour milk* is still a surprisingly common staple food, except in Portuguese territories. Whether this applies equally to women and children is doubtful. *Stamp mealies* are made by pounding mealies with a wooden "pestle" in a hollow tree trunk, the grain being broken to about the size of a pea and some of the chaff removed. *Stone ground mealie meal* is made by placing the corn on a flat stone and rolling it with a rounded one, some of the chaff usually being removed. *Beans* refer to a native variety. *Spinach*, made by boiling the tender growing shoots of a large variety of wild and cultivated plants, is seldom eaten by the men of the Transkei tribes, who regard it as effeminate; it is presumed that their women do eat it. The Basutos, Bechuanas and Portuguese natives have none of this prejudice and "spinach" forms an important item in their diets. It is a common practice to dry out and store "spinach" for winter use. *Peanuts* of a native variety are cultivated by Portuguese natives, Transkei natives obtaining them at the trading stores.

A comparison of the food eaten by different tribes and in different districts (1) showed that the Xosa, Pondo and Fingo tribes, i.e. the natives coming from the Transkei, are similar in their feeding and that they differ more or less widely from the others and, because it was considered desirable to make the comparison within a homogeneous group, the subsequent analysis was confined to the Transkei group of 609 cases. None of the other areas had supplied enough cases to make a second analysis in another area worthwhile. The 609 Transkei natives were grouped afresh for each of their common foodstuffs. At each analysis three groups were compiled: 1. those who never ate that particular foodstuff (0), 2. those who at it occasionally (+), 3. those who ate it regularly (several times a week) (++). The number of cases in each of these three groups was counted; then, by a further analysis the number of carious mouths and the number of carious teeth in each group were counted. The figures in the fourth, fifth and sixth columns were calculated from the first three columns, except in those groups which contained less than forty cases (an arbitrary figure, below which it was not thought that a quotient would be at all reliable). In dealing with some of the native foods a correction was found to be necessary because of regular asso. iation with European foods. Fresh milk is seldom taken with anything but tea and coffee, so that if, as at first appears, there is an increase of caries with an increased consumption of fresh milk, this is not likely to be due to the milk *per se*; this apparent association disappears when all cases taking regular European foods are eliminated from the calculation. Similarly those who eat potatoes, kaffir corn and peanuts regularly must buy them from shops where they would be likely to buy European

foods as well. It is those males who have overcome tribal prejudices and given up primitive ways of life who eat "spinach" as a regular article of food. In each of these instances, as the tables show, exclusion of all those cases marked ++ for one or more of the European foods removes most of those marked ++ for fresh milk, potatoes, kaffir corn, peanuts and "spinach".

RESULTS

Between nine-hundred and a thousand new recruits coming from Transkei, Portuguese East Africa, Bechuanaland, Bastuoland and Nyasaland were examined. The number of cases, of carious mouths and of caries teeth from the different areas are summarized in Table I. A more detailed discussion of the natives' teeth is given elsewhere

TABLE I

	NO. CASES	NO. CARIOUS CASES	NO. CARIOUS TEETH	PERCENT. CARIOUS CASES	NO. CARIOUS TEETH PER CASE	NO. CARIOUS TEETH PER CARIOUS CASE
Area						
Transkei	609	249	768	41	1.3	3.0
Port. E. Africa	198	103	376	52	1.9	3.6
Bechuanaland	25	10	26	40	1.0	2.6
Basutoland	21	5	21	24	1.0	4.4
Nyasaland	86	24	75	28	0.9	3.1
Tribe						
Xosa	411	169	494	41	1.2	3.0
Pondo	113	45	158	40	1.4	3.5
Fingo	76	36	106	47	1.4	2.9
Shangaan	176	86	300	49	1.7	3.5
Machopi	20	16	68	80	3.4	4.2

(1), but certain incidental observations may be of interest. There are no macroscopic differences between the enamel of Europeans and Bantu. Some of the irregularities met with in the Bantu are (a) the anterior portions of many jaws are cramped, generally with displacement of the second incisors, (b) a marked diastema is sometimes found between the first incisors, sometimes in both maxilla and mandible, sometimes in the maxilla alone, (c) there are many supernumerary teeth, (d) pigmented enamel is found, (e) there are many cases of accidentally fractured teeth. There are, on the whole, very few cases of unerupted or missing teeth. The practice of filing the incisors is

TABLE II

		NO. CASES	NO. CARIOUS MOUTHS	NO. CARIOUS TEETH	PERCENT. CARIOUS MOUTHS	NO. CARIOUS TEETH PER CASE	NO. CARIOUS TEETH PER CARIOUS MOUTH
Native Foods							
Fresh milk	0	339	140	420	41	1.2	3.0
	+	153	54	160	35	1.0	2.9
	++	117	59	188	51	1.6	3.2
Fresh milk and no European foods ++	0	218	94	284	43	1.3	3.0
	+	88	29	86	33	1.0	3.0
	++	42	18	59	43	1.4	3.3
Sour milk	0	33	14	45	—	—	—
	+	122	53	148	44	1.2	2.8
	++	454	186	575	41	1.3	3.1
Whole mealies	0	74	30	98	40	1.3	3.3
	+	340	137	405	40	1.2	3.0
	++	195	86	265	44	1.4	3.1
Stamp mealies	0	22	9	26	—	—	—
	+	244	106	322	44	1.3	3.0
	++	343	138	420	40	1.2	3.0
Stone ground mealie meal	0	61	25	80	41	1.3	3.2
	+	366	153	470	42	1.3	3.1
	++	182	75	218	41	1.2	2.9
Kaffir corn	0	172	68	232	40	1.3	3.4
	+	371	151	446	41	1.2	3.0
	++	66	34	90	51	1.4	2.6
Kaffir corn and no European foods ++	0	109	43	150	39	1.4	3.5
	+	210	88	258	42	1.2	2.9
	++	29	10	21	—	—	—
Beans	0	39	18	65	—	—	—
	+	316	132	384	42	1.2	2.9
	++	254	103	319	40	1.3	3.1
Meat	0	7	4	22	—	—	—
	+	502	209	610	41	1.1	3.0
	++	100	40	136	40	1.4	3.4
Potatoes	0	213	87	263	41	1.2	3.0
	+	326	133	381	41	1.2	2.9
	++	70	33	124	47	1.8	3.8
Potatoes and no European foods ++	0	160	60	181	38	1.1	3.0
	+	170	75	223	44	1.3	3.0
	++	18	6	25	—	—	—

TABLE II—*Continued*

		NO. CASES	NO. CARIOUS MOUTHS	NO. CARIOUS TEETH	PERCENT. CARIOUS MOUTHS	NO. CARIOUS TEETH PER CASE	NO. CARIOUS TEETH PER CARIOUS MOUTH
Native Foods—Cont.							
Sweet Potatoes	0	309	116	373	38	1.2	3.2
	+	199	91	247	46	1.2	2.7
	++	101	46	148	45	1.5	3.2
"Spinach"	0	536	221	690	41	1.3	3.1
	+	54	21	44	37	0.8	2.2
	++	19	11	34	—	—	—
"Spinach" and no European foods	0	315	130	410	41	1.3	3.2
	+	28	10	16	—	—	—
	++	5	1	3	—	—	—
Peanuts	0	471	193	603	41	1.3	3.1
	+	119	47	108	39	0.9	2.3
	++	19	13	57	—	—	—
Peanuts and no European foods	0	295	122	394	41	1.3	3.2
	+	53	19	35	36	0.7	1.8
	++	0	0	0	—	—	—
Kaffir beer	0	40	18	74	45	1.9	4.1
	+	260	113	326	43	1.3	2.9
	++	309	122	368	40	1.2	3.0
Pumpkin	0	24	12	53	—	—	—
	+	249	111	332	45	1.3	3.0
	++	336	130	383	39	1.1	2.9
European Foods							
Machine ground mealie meal	0	244	93	290	38	1.2	3.1
	+	295	122	358	42	1.3	2.9
	++	70	38	120	54	1.7	3.2
European bread	0	193	84	274	43	1.4	3.3
	+	362	144	407	40	1.1	2.8
	++	44	25	87	57	2.0	3.5
Sugar	0	85	34	117	40	1.4	3.4
	+	307	120	341	39	1.1	2.8
	++	217	99	310	46	1.4	3.1
Sweets	0	72	27	110	37	1.5	4.1
	+	491	198	562	40	1.1	2.8
	++	46	28	96	61	2.1	3.4

TABLE II—*Concluded*

		NO. CASES	NO. CARIOUS MOUTHS	NO. CARIOUS TEETH	PERCENT. CARIOUS MOUTHS	NO. CARIOUS TEETH PER CASE	NO. CARIOUS TEETH PER CARIOUS MOUTH
European Foods—Cont.							
Jam	0	304	114	366	38	1.2	3.2
	+	274	121	343	44	1.3	2.8
	++	31	18	59	—	—	—
Tea	0	163	72	233	44	1.4	3.2
	+	258	93	262	36	1.0	2.8
	++	188	88	273	47	1.5	3.1
Coffee	0	188	76	233	41	1.2	3.1
	+	281	110	323	39	1.1	2.9
	++	140	67	212	48	1.5	3.2

still fairly common among the Machopi, and also, we are informed, among the Tongas. In the kraals visited marginal gingivitis and periodontal disease are rare, but they occur more frequently among the natives examined at the Johannesburg compound. Although it is certain that many of the cases examined were syphilitic or had subacute scurvy, there is no marked evidence of these conditions in the mouths examined. The average Bantu has a very large tongue.

Analysis of Data from Transkei Cases. Tables II and III show the association of dental caries with individual items of the diet in the Transkei natives. Occasional natives spoke of eating foodstuffs not mentioned in these tables, but, for all practical purposes, they may be regarded as a full list of the foods in common use among the Bantu concerned.

With the material at their disposal, the authors obviously could not set out to give an accurate quantitative measure of the correlation. They do not regard their results as doing more than indicating, in a qualitative manner, where the principal cause of this decay is likely to be found.

The number of carious teeth. The figures in the *fifth column* (Tables II and III) are useful in giving support to, or casting a doubt upon, the conclusions drawn from the *fourth* column. Where there are large differences between these columns, the explanation of these differ-

ences is obscure. The number of carious teeth per carious mouth (*sixth column*) is the least informative and this agrees with the previous investigation (2).

The effect of milk. So few of the natives in this group took no sour milk that the effect of taking sour milk cannot be determined with certainty. However, the Portuguese natives, who do not get as much sour milk as the Transkei natives (1), have not only a greater percentage of carious mouths but also more carious teeth per mouth. It can only be said in this instance that a greater incidence of caries is associated with a diminished milk intake; there are so many other differences between the natives from these two areas that it would be unjustifiable to claim dogmatically that the low milk supply is the one difference which causes the higher incidence of caries among the Portuguese natives.

The number of carious mouths. The best indication of the influence of the diet on the teeth is to be derived from the *fourth column*, the percentage of carious mouths in each group. Table II shows that, among native foods, regular consumption of fresh milk, kaffir corn, potatoes and sweet potatoes is associated with an increased percentage of carious mouths. From the first two columns, it appears that ++ peanuts and ++ "spinach" are also associated with increased carious cases, although in these groups the number is too few to justify calculating a percentage. As all these native foods except sweet potatoes are frequently associated with European foods, if all cases eating any article of European food regularly are excluded, most or all of the regular eaters of each of these foods are eliminated as well. The cases that are left show no such association. As far as the native foods are concerned, though several seem on casual examination to be associated with an increased proportion of carious cases, a more searching analysis shows that only sweet potatoes retain this association if the cases having a ++ for any European food, are excluded.

Table II shows that the position is different when European foods are studied. Taken item by item, regular machine ground mealie meal, European bread, sugar, sweets, jam, tea and coffee are all found to be associated with a percentage of carious mouths above the average and well above the groups eating none of each of these foods. This agrees with and amplifies the results obtained in the previous work

(2). The fact that the figure for ++ sugar is not very much above the average may be due to the fact that most of the cases in this series took sugar in their tea or coffee only. When the three sugary foods are taken together, the association is much more definite (Table III).

Effect of erroneous answers. While the authors are convinced that the information they were given was reliable in almost all cases, it is important to discover what the result of erroneous answers would be.

TABLE III

FOODS CONTAINING SUGAR, i.e. SUGAR, SWEETS AND JAM	NO. CASES	NO. CARIOUS MOUTHS	NO. CARIOUS TEETH	PERCENT. CARIOUS MOUTHS	NO. CARIOUS TEETH PER CASE	NO. CARIOUS TEETH PER CARIOUS MOUTH
None of them eaten regularly.....	381	149	447	39	1.2	3.0
One eaten regularly.....	178	75	221	42	1.2	3.0
Two eaten regularly.....	32	16	56	58	2.1	3.6
Three eaten regularly.....	18	13	48			
GIVING 0 POINTS FOR NEVER, 1 FOR OCCASIONALLY AND 2 FOR REGULARLY—						
GROUP HAVING TOTAL OF						
0 Points	23	7	23	36	1.3	3.6
1 Point	68	26	96			
2 Points	146	58	190	40	1.1	2.9
3 Points	244	96	254			
4 Points	86	42	128	52	1.6	3.1
5 Points	25	12	33			
6 Points	17	12	44			

The number of those giving false answers should be distributed evenly among the carious and non-carious groups by the law of averages, at least in large groups. Where the carious group is numerically equal to the non-carious this would result in zero percentage error and where the two groups are nearly equal in only slight percentage error. Moreover, the false answers would not produce fictitious correlations but would reduce the degree of the correlation already existing.

DISCUSSION

It is not suggested that European food causes all the caries in the Bantu. It is difficult to estimate what percentage of absolutely untouched kraal natives have caries, because there are so few left, but it is probably about twenty or thirty per cent. In 40 prehistoric (i.e. pre-European) Bantu skulls[3] there were 749 teeth, 31 of which were decayed. In the native way of living itself, there are some minor causes of caries. This paper does not deal with those, but with the agency in the civilized way of living which causes an increase in caries. The twenty or thirty per cent of carious cases in primitive natives is a minor matter compared with the rampant caries in civilized communities.

Sugar and/or refined cereals. This theory of decay is the one that obviously suggests itself as the explanation of our findings. It has been shown that the increased incidence of caries is associated with the consumption of certain European foods. This does not necessarily imply that it is caused by the eating of these foods. There is, however, nothing that the native does when he begins to become civilized, which can be advanced as reasonably as an explanation, as his change in diet. The European foods listed are those he takes to first, and it is among them that the cause of his dental deterioration is likely to be found. It is unnecessary to suppose that they all play a part. Tea and coffee can be associated with increased decay only because they are associated with the eating of bread, machine-ground mealie meal and sugar. One is left with the conclusion, that one or more of the three following factors probably cause the increase in caries in those natives taking to a European manner of living: the eating of (a) European bread (almost always a white bread), (b) machine ground mealie meal, (c) foods containing sugar. "One or more" is said advisedly; one or two of the factors may be merely associated with the real cause.

It is possible that cereal grains *per se* contribute to the decay of the native teeth. The South African Bantu have lived on a high cereal diet since time immemorial and it is possible that the few decayed teeth in the primitive and pre-historic Bantu are caused by the cereals.

[3] Anatomy Department, University of Witwatersrand.

But the natives do not take more cereals when they become Europeanised, so that cereals *per se* are not the cause of the accelerated decay considered in this paper. For equally good reasons, it may be argued that sugar, as it occurs in nature, is not a major cause. Natives in the sugar-cane fields and factories chew cane regularly, and yet, neither in this nor in other countries have they been found to be more prone to caries than their fellows. Fruits contain sugar and they have never fallen under suspicion as producers of caries. Of the three factors, two have this in common—they are refined. The case of mealie meal is more difficult to decide. The native "stone ground mealie meal" (which is not associated with increased caries) is usually refined to a limited degree by winnowing; on the other hand, machine ground mealie meal as sold to the natives in the territories is usually milled on simple plate mills and is seldom subjected to much sifting. It cannot be said that machine ground mealie meal is much, if at all, more refined than the natives' own product.

If machine ground mealie meal is the real cause or one of the real causes of caries in these cases, how it causes caries is still obscure. If however, either white bread or refined sugar or both contribute to the increased decay, a feasible explanation offers itself. Carbohydrate undergoes an acid fermentation if it remains in the mouth: the well-known chemico-parasitic theory of decay being based on this knowledge. That theory does not explain, however, why some forms of carbohydrates—potatoes and crude cane sugar—produce no caries, while other forms—wheat flour or refined cane sugar—do. It is unlikely that one form produces a less acid medium than the others, preliminary experiments by the authors suggesting that refined and crude carbohydrates (e.g. refined white flour and whole wheat meal, refined sugar and crude cane juice) reach the same degree of acidity when incubated with saliva. The authors therefore suggest (a) that, in the natural or crude state, the carbohydrate is accompanied by some protective agent, (b) that refinement removes this protective agent. The authors do not wish, at this stage, to advance suggestions as to what this protective agency may be.

SUMMARY

1. The distribution of caries among the Bantu in different areas and of different tribes in Southern Africa is recorded. 2. In 609 cases

from the Transkei area the incidence of caries is correlated with different items in the diet. 3. With the possible exception of sweet potatoes, none of the items in the traditional native diet shows a correlation, either positive or negative, with an increased percentage of carious cases. 4. Certain conclusions from a previous paper are confirmed, viz. that there is a greater incidence of caries in those natives who have taken to eating European foods. 5. The chemico-parasitic theory may serve, with certain modifications and extensions to explain this. It is suggested that one or more of the following foods *cause* the increased incidence of caries among those who eat them regularly: a. machine ground mealie meal, b. white bread, c. refined sugar or foods containing it. 6. If either white bread or foods containing sugar or both are real causes, an explanation of how they produce their effect is tentatively advanced. It is suggested that the carbohydrates of sugar cane and/or wheat are accompanied, in the natural or crude state, by a "protective agent"; and that refined sugar and/or refined wheat flour cause decay as a result of removal of this "protective agent" in the process of refining.

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A HISTOPATHOLOGIC AND RADIOGRAPHIC STUDY OF THE TEETH OF DOGS FED A RACHITOGENIC DIET[1]

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Observations on the dentitions of animals fed experimental diets have established the importance of calcium and phosphorus metabolism in the development and maintenance of the dental tissues. Until recently, the hard tissues of the teeth have been considered stable substances. There was no known relationship between metabolic activity and the variation in the composition and structure of fully formed teeth. By varying any one or more of the factors which influenced the calcium-phosphorus metabolism, we were able to bring about an abstraction of calcium from the teeth of animals which we are led to believe precedes dental caries. In the early stages of these experiments minute examination revealed no abnormalities on the surfaces of the teeth. The decalcification was apparent only radiographically, radiolucent areas being observed which were not unlike the lesions found in incipient human caries. On prolongation of the experiment there occurs active destruction of the enamel and dentin. Routine x-ray examinations of human mouths have often disclosed similar radiolucent areas in the teeth while subsequent operative procedures often fail to disclose any lesions on the tooth surfaces. Clinical experience has shown that if these radiolucent areas are not excavated dental decay usually ensues. Recently, Fish (1) analyzed teeth of dogs showing parathyroid abnormality and found a 14.93% loss of calcium in the teeth in one animal and 6.76% loss in another.

In previous communications (2, 3, 4) we have described the lesions produced by calcium starvation and calcium mobilization. These

[1] Acknowledgment is hereby made of the financial assistance of the Commonwealth Fund in the pursuit of this study.

changes occur in the pulp, dentin and enamel. The pulpal pathology invariably preceded the carious lesions on the surfaces of the teeth in our animals. These incipient phenomena are important in the ultimate production of radiolucency and true caries. Where visible lesions are produced the radiolucency always extends beyond the area of destruction. Calcium and phosphorus analyses were made at regular intervals. The extent of tooth abnormality and destruction varies with the duration of the experiment and condition of the animal. Administration of excessive amounts of viosterol accompanying the abnormal Ca-P ratio in the diet, was found to have accelerated the process. All autopsy material was studied microscopically.

Four eight week old puppies from the same litter (nos. 301, 302, 303, 304), were placed on the same rachitogenic diet (4). Animal 304 received in addition, 8 drops of viosterol (250 D) daily. After being on the prescribed diet for 24 days, 301 developed a characteristic gait. Five days later muscular weakness and painful joints were noticed, x-rays of the joints showing definite rickets. In 37 days 302 developed the characteristic rachitic gait. After 82 days of feeding 303 was unable to walk. Pup 304 receiving viosterol, remained apparently normal, clinically. Since the diet was deficient in calcium, although compensated by the presence of viosterol, he was the control to the group but not a true normal. Animal 301 was sacrificed at 130 days of experimental feeding, 302 at 170 days and 303 and 304 at 190 days. The lesions observed in this group were identical with those found in our earlier experiments in 93 rachitic animals (9 dogs and 84 rats).

The pulps of all the deficient animals exhibited similar pathosis. All stages of odontoblastic degeneration and even complete absence of these cells were observed in the teeth. The incipient odontoblastic lesion is a distortion of the usual position of these cells. Under low power, the appearance is quite normal but higher enlargement of the odontoblastic layer reveals the beginning of the degenerative process. The contours of the cells vary from columnar to ovoid; the nuclei lose their distinctness; the number of nucleoli is reduced and the nuclear membrane gradually merges with the degenerated cytoplasm of the cell. The variation in staining of the nuclei undergoing karyolysis and the spherical outlines formed by the cell walls may give the appearance of a marked infiltration of phagocytic cells. Pyknosis is also found and is combined with the concurrent cytoplasmic degeneration presenting the characteristic appearance of "foam-cells." The initial stage of perinuclear destruction is a honeycombing of the cytoplasm, small vacuoles soon appear distributed throughout the cell

body and subsequent merging of these areas produces the "foam-cell" appearance seen in many specimens (*fig. 2B*). Fat and mucous stains were negative. Hematoxylin and eosin and trichrome staining exposed a delicate mesh of strands passing from the region of the nucleus towards the cell wall. The areas included within the reticulum do not stain. The staining reactions and the microscopic appearance indicate that the cells have undergone a serous degeneration, the cytoplasm being exposed as a delicate reticulum. The dentin wall presents a scalloped appearance in which these intracellular vacuoles lie (*fig. 3A*).

Concurrent with the above phenomena there is a proliferation of the reticular cells (fibroblasts) of the pulp immediately beneath the odontoblastic zone (*fig. 5C*). As the process advances, these connective tissue cells force their way between the degenerated odontoblasts and become attached to the dentin wall. The removal of the odontoblasts by an autolytic process facilitates their replacement by connective tissue cells (*fig. 4B*). The blood and lymph vessels are markedly distended. The vascularity and connective tissue proliferation observed beneath the odontoblasts are reflected throughout the entire pulp. The delicate reticulum of the pulp, classically compared with Wharton's jelly, becomes a mass of compact connective tissue. This fibrosis is accompanied by a marked increase in the collagen content. The destruction of the odontoblasts facilitates the demonstration of the organic matrix fibres of the dentin. These von Korff's fibers stained readily with silver and can be traced for quite a distance into the substance of the pulp. Accompanying this complex picture is a radiolucency (*fig. 7A*) in the crowns of these teeth invariably observed on prolongation of the experiment.

Odontoblastic destruction occurs comparatively early in the experiment. Low power studies do not show any gross variation from the normal, the cells being regularly arranged along the periphery of the pulp and the supporting tissue somewhat more dense beneath the odontoblastic layer. A high power study of a portion of the same field reveals the inception of the pathologic process discussed. Many nuclei stain clearly and retain their normal positions; others are becoming fainter. A stippling of the cytoplasm and subsequent honeycombing is noticeable. At this stage, the cell outlines are unaffected.

A more advanced degenerative process is seen in *fig. 1*. Vacuoles have developed in the coarse cytoplasmic honey-combing; the nuclei are becoming more indistinct; the cell walls are increasingly irregular merging one cell with another where the destruction is complete. A few odontoblasts still retain their outlines although the cytoplasm is markedly altered. No fibrosis is apparent. There seems to be a definite accellular zone between the degenerating odontoblasts and the connective tissue of the pulp. The "foam-cell" appearance of the degenerating odontoblasts is shown in *fig. 2*. The nuclei seem hazy and are eccentrically placed, there are fine cytoplasmic strands extending somewhat radially from the region of the nuclei. The cell outlines are indefinite in many areas. One cell in the field seems to contain two nuclei, one undergoing lysis, the other, pyknotic changes and fine cytoplasmic strands seem to connect both nuclei. The autolytic process is well advanced. There is a proliferation of the underlying fibroblasts. The latter have lost their stellate appearance and are spindle shaped.

Fig. 3 demonstrates the appearance of the pulp prior to complete odontoblastic disappearance. Large cystic areas appear in juxtaposition to the dentin wall and rest in concavities in the predentin. Some nuclear remains can be seen. The intricate mesh can be observed in the odontoblasts. There is a marked proliferation of connective tissue cells beneath the odontoblastic layer, and they are forcing their way between the degenerated odontoblasts to become attached to the predentin. There is a proliferation of blood vessels in this zone. The organic matrix of the dentin is readily visible in higher power studies of these specimens (*fig. 3*). The detin wall is very irregular (*fig. 4*). There is a complete absence of odontoblasts in this later stage. A marked condensation of connective tissue is seen at the periphery of the pulp. The proximity of the nuclei gives the appearance of odontoblasts cut tangentially. The more internal portions of the pulp are not greatly affected. The distended blood vessels can be seen immediately beneath the zone of cellular condensation. High power studies of the irregularities observed in the dentin wall often reveal fine capillaries extending into the dentin proper and at times larger vessels engorged with red blood cells can be seen. There is a marked condensation of the peripheral connective tissue cells. The

organic matrix of the dentin is discernible in all staining, especially with Laidlaw's silver stain for connective tissue (8). The von Korf's fibers can be seen extending far into the pulp. The relationship between odontoblastic degeneration and underlying fibrotic metamorphosis can be seen in *fig. 5*. The odontoblasts still retain their form although pathologic changes are evident. There is marked condensation of the connective tissue cells beneath the odontoblasts. The apparent fibrosis in the depth of the pulp is really a fibrosis adjacent to the opposite dentin wall. In some teeth where the pathologic process is greatly advanced, degeneration of the entire pulp occurs.

The pulpal phenomena discussed above are unquestionably significant in the production of radiolucent areas in the crown of teeth whose surfaces appear perfectly normal. *Fig. 6* demonstrates the clinical appearance of such teeth. The enamel is intact and shows no discoloration. The roentgenograms (*fig. 7A*) reveal the extensive radio lucency. The enamel can be followed completely around the tooth. In some regions it is markedly decalcified. The mandibular canine presents a large radiolucent zone on the distal aspect. The lesions in the maxillary canine are more diffuse.

Histologists have been conscious of the rôle played by odontoblasts in the calcification of the tooth. Becks and Ryder (5), in studying the incisors of rats on rachitogenic diets, found that in areas where the ondotoblasts have atrophied there is a cessation of calcification; but in the adjacent parts where the cells were normal calcification was complete. In the erupted teeth of dogs a decalcification is produced by a reversal of the calcifying process. These decalcified zones become carious. There is an unusual vascularity in the pulps of these animals. Studies in bone have demonstrated that there is a cessation of calcification and a tendency towards resorption when the blood supply is abundant (6, 7). Considering the structural similarity between dentin and bone, a like relationship between the odontoblastic degeneration and increased vascularity, and decalcification of the teeth is apparent.

SUMMARY

Variations in the calcium phosphorus metabolism have produced consistent changes in the teeth of experimental animals. The first

noticeable lesion is in the odontoblastic layer accompanied by pulpal engorgement. These cells undergo a serous degeneration and subsequent autolysis. There is a proliferation of the underlying fibroblasts which replace the odontoblasts. The pulpal tissue undergoes fibrosis. Combined with these phenomena is a marked increase in the vascularity of the tissue. Prolongation of the time of experimental feeding produces an unquestionable radiolucency in the crowns of the teeth. In instances where the animals were kept alive for long periods, caries-like lesions developed in these areas.

We wish to thank Dr. C. C. Lieb for his helpful guidance in the planning and conducting of these experiments, and Dr. C. F. Bödecker for his valuable aid in the interpretation of the dental findings. Our thanks are due Dr. E. Applebaum for the photomicrography.

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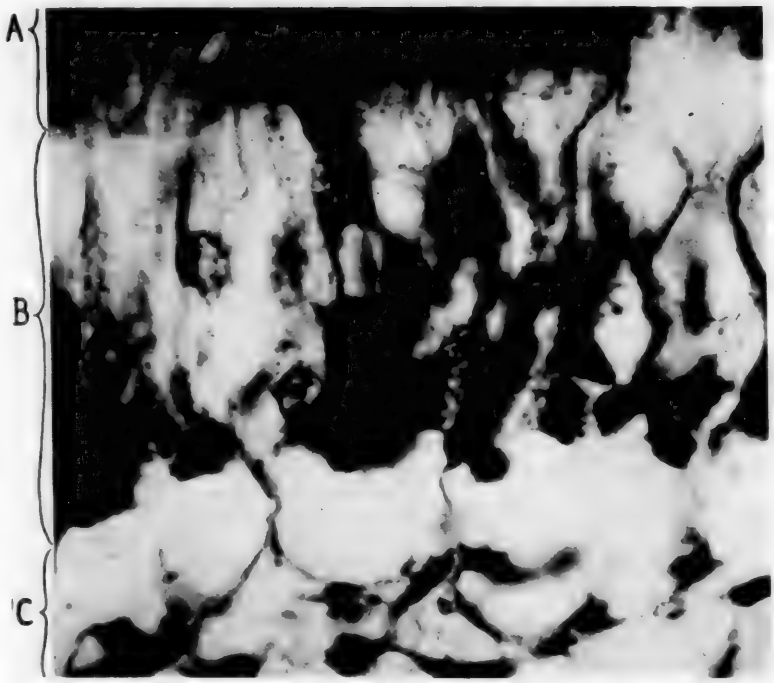

FIG. 1. Advanced stage in odontoblastic degeneration. Nuclei are becoming less distinct. Cytoplasm degeneration is well advanced. Note demarcation between odontoblasts and reticular cells. A, dentin. B, odontoblasts. C, reticular cells. 2 mm. obj., 4 ocular. Orig. mag.—2000 ×. Bellows length—90 cm. Paraffin section, hematoxylin and eosin stain.

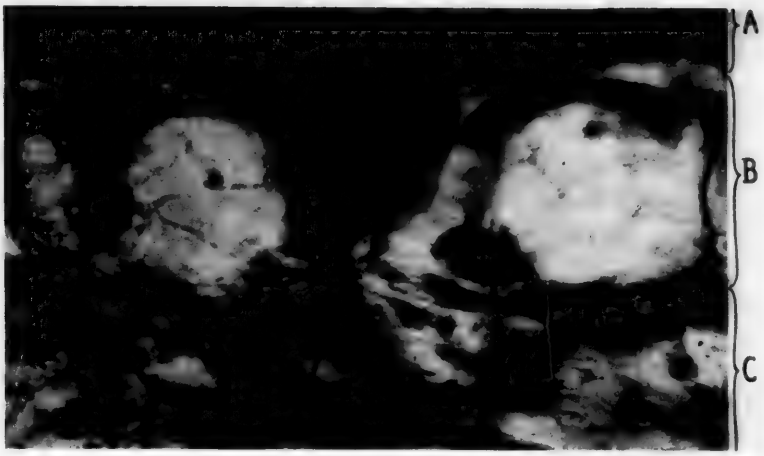

FIG. 2. Serous degeneration of odontoblasts. Note fine fibrillae emanating from nucleus. One area shows two nuclei within the same cell wall. Underlying connective tissue has undergone fibrotic changes. A, dentin. B, degenerative odontoblasts. C, reticular cells. 2 mm. obj., 4 ocular. Orig. mag.—2000 ×. Bellows length—90 cm. Celloidin section, Masson stain.

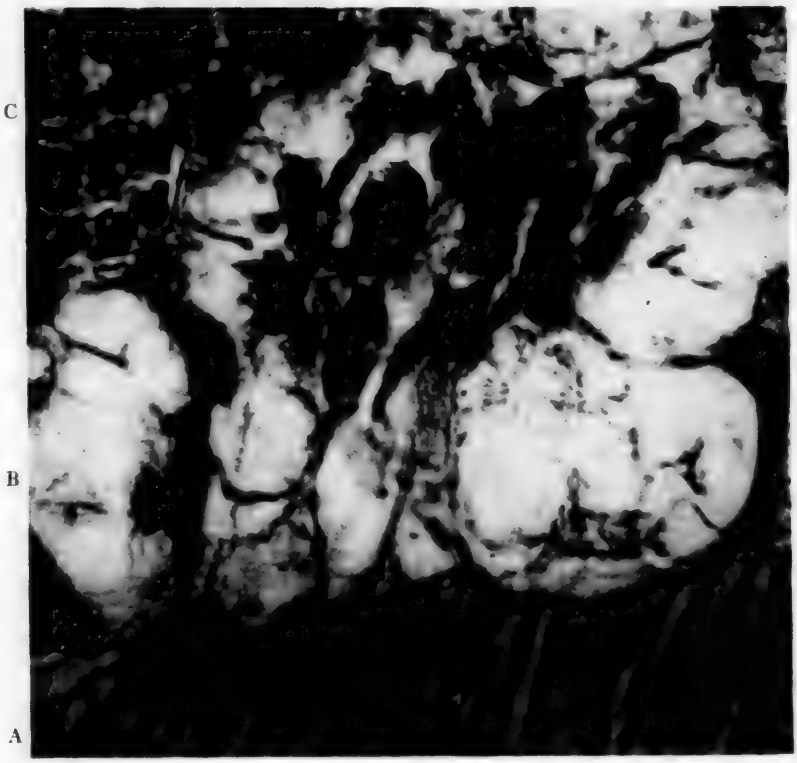

FIG. 3. Final stage in odontoblastic degeneration. Large cystic areas contain nuclear remains and fibrinous network. Areas containing groups of nuclei may be proliferating connective tissue cells forcing their way towards the dentin wall. A, dentin. B, degenerate odontoblasts. C, connective tissue cells. 2 mm. obj., 4 ocular. Orig. mag.—2000 ×. Bellows length—90 cm. Celloidin section, Masson stain.

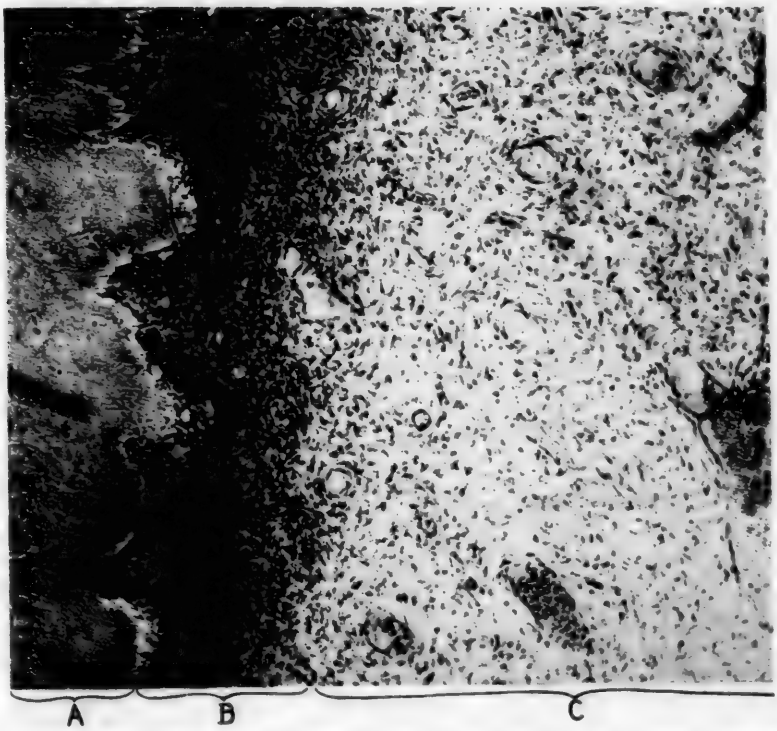

FIG. 4. Note absence of odontoblasts. Connective tissue cells are closely packed along irregular dentin wall. Blood vessels distended. A, dentin. B, dense layer of connective tissue cells. C, underlying pulpal tissue. 16 mm. obj., 4 ocular. Orig. mag.—200 ×. Bellows length ~90 cm. Celloidin section, Masson stain.

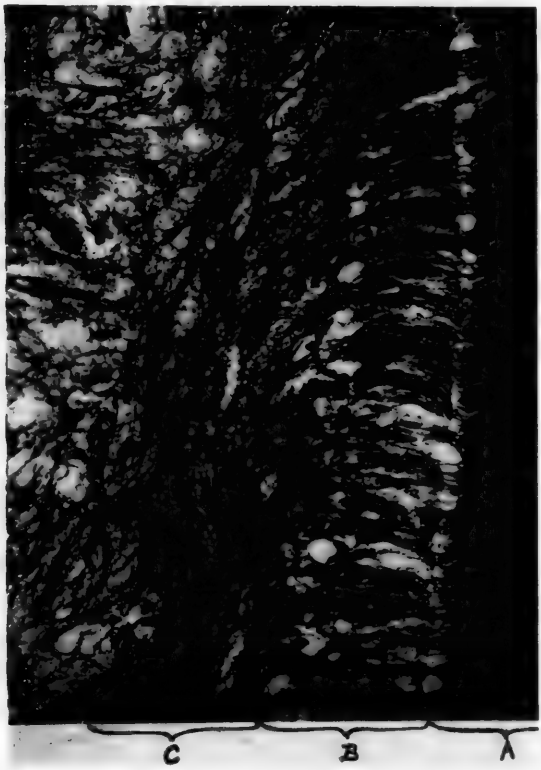

FIG. 5

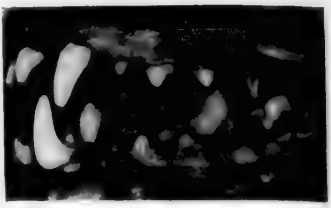

FIG. 6

FIG. 7

FIG. 5. Pulp Horn. Simultaneous degeneration and underlying fibrosis. A, dentin. B, odontoblasts. C, peripheral fibrosis. 8 mm. obj., 4 ocular. Orig. mag.—750 ×. Bellows length—90 cm. Paraffin section, hematoxylin and eosin stain.

FIG. 6. Photograph of jaws of dog. Canines are perfectly normal in *appearance*. Observe large deposits of calculus on molars and premolars.

FIG. 7. A. Maxillary canine (from animal in *fig. 6*) shows a diffuse radiolucency. The mandicular canine presents a sharply outlined radiolucency on the distal aspect. Note decalcification of enamel, not seen clinically. B. Canine of control animal (radiograph).

THE AVERAGE MICROHARDNESS OF APATITE

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In a previous paper (1), hardness studies of samples of the various minerals composing the Mohs' scale included a measurement on an apatite specimen with an average microhardness of 500. Since similar measurements of certain dry, polished tooth enamel surfaces were found (2) to give average microhardnesses of from 1300 to 1800 and since the mineral phase of enamel (3) is similar chemically and crystallographically to apatite, the question arose as to the validity of the microhardness values found for the apatite crystal. Three courses were pursued: (1) the microcuts on the above-mentioned apatite crystal were studied carefully and a new series of 10 microcuts on that crystal were measured; (2) the microhardnesses of a series of 11 apatite specimens were obtained; and (3) five "perfect" crystals of apatite were studied with great care to determine the average magnitude of microhardness, the variation (if any) in the hardness of the different types of faces, and the hardness difference (if any) produced by cutting in different directions on a given face.

The microcuts on the first-mentioned apatite crystal were found to show a marked "chattering" effect although the edges were not shattered and the widths were quite uniform. The new series of ten cuts made on two faces (in pairs with opposite directions of cutting) were all uniformly smooth. Consequently, it is inferred that the diamond previously used was defective. Seven readings per cut on the new cuts gave fairly uniform average widths varying from 2.4 to 2.7 microns and average microhardnesses ranged from 1500 to 1600. In Table I are given the average microhardnesses of the series of 11 specimens of apatite crystals collected from various sources.[1] Eight to eighteen

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[1] Obtained from Ward's Natural Science Establishment, Rochester, N. Y.

readings were made on the various cuts, giving average widths of 2.4 to 3.4 microns and average microhardnesses ranging from 870 to 1740.

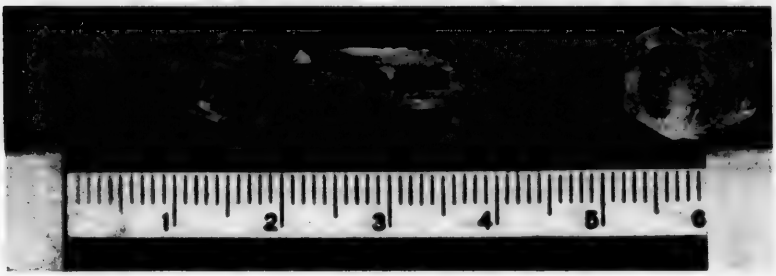

FIG. 1. Size and shape of five "perfect crystals (compare with mm. scale below). Left to right: 100 (Source-Ehren friedersdorf, Saxony), 101 (Jumilla, Spain), 102 (Durango, Mexico), 103 (Halmberget, Gellivari, Sweden), 104 (St. Gothard, Switzerland).

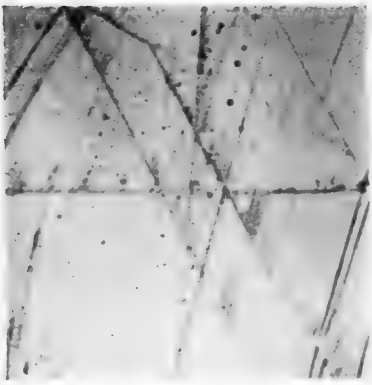

FIG. 2

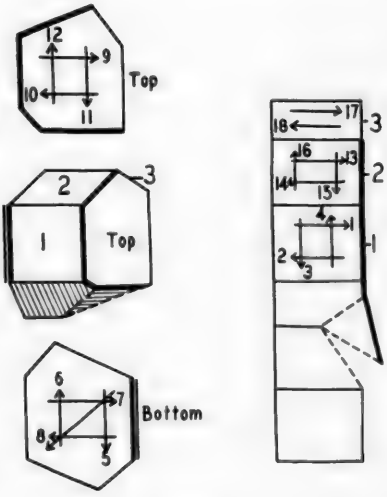

FIG. 3

FIG. 2. The intersection of two microcuts on the surface (Face No. 4) of crystal No. 104 (compare *fig. 5*). Original magnification 550 times.

FIG. 3. Crystal 100. Directions of microcuts may be compared from surface to surface. Diagonal cut on "Bottom" face made using microcharacter at Lumen Bearing Corporation.

In *fig. 1* are shown the five "perfect" crystals of apatites selected for intensive study. The small size of the crystals and the number of determinations made on each of several faces (see subsequent figures) illustrate the applicability of the microcharacter to mineral specimens. *Fig. 2* shows the appearance of two intersecting microcuts on a face (Face No. 4, *fig. 7*) of crystal No. 104. *Figs. 3 to 7* show the various faces on the five crystals which were measured and also the direction of microcuts on each face. The relation of the direction and placing of each cut relative to the principal crystal axes may be reconstructed from the drawings.

TABLE I

APATITE SAMPLE NO.	SOURCE	NO. READINGS	AV. WIDTH (μ)	AV. MICRO-HARDNESS
1	Haddon Neck, Conn.	8	2.6	1480
2	Murcia, Spain	16	2.9	1190
3	Franklin, N. J.	12	2.8	1280
4	Ottawa Valley, Ont.	15	2.4	1740
5	Hastings County, Ont.	18	2.7	1370[1]
6	St. Gothard, Switzerland	12	2.6	1480
11	Tray Hill, Ont.	14	2.4	1740
20	Battle Lake, Canada	16	2.6	1480
21	Durango, Mexico	12	2.6	1480
22	Simotsuko, Japan	9	2.9	1190
29	Auburn, Maine	14	3.4	870
40	Mesa Grande, Calif.	15	2.6	1480

[1] This value checked independently by another microcharacter gave an average microhardness of 1030.

Five faces were studied on crystal No. 100; of these, two were type 0001 (Miller indices), three were type $10\bar{1}0$. The microhardnesses averaged about 1280. Six faces were studied on crystal No. 101, three of the type $10\bar{1}1$ and three of the type $10\bar{1}0$. An average microhardness of 1190 was found. Three faces were studied on crystal No. 102, one being type $10\bar{1}0$, one of type $11\bar{2}0$ and the third of type $10\bar{1}1$. The average microhardness was 1110. Six faces were studied on crystal No. 103, all of the type $10\bar{1}0$. The microhardness ranged from 1110 to 1190. Five faces were studied on crystal No. 104, of which one was of type 0001, three of type $10\bar{1}0$, and one of type $11\bar{2}1$. The microhardness ranged from 1190 to 1280.

The identifications of the faces on crystals Nos. 102 and 104 were made from goniometer measurements by Robert D. Butler at the Massachusetts Institute of Technology.

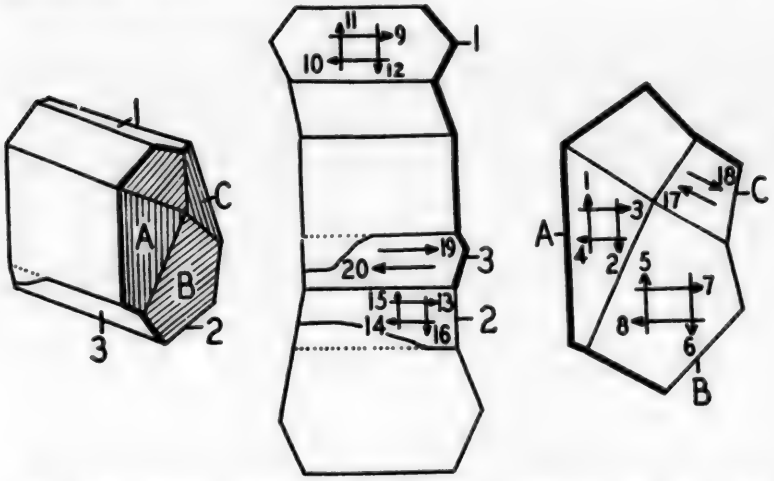

FIG. 4. Crystal 101. Directions of paired microcuts relative to crystal axes may be noted.

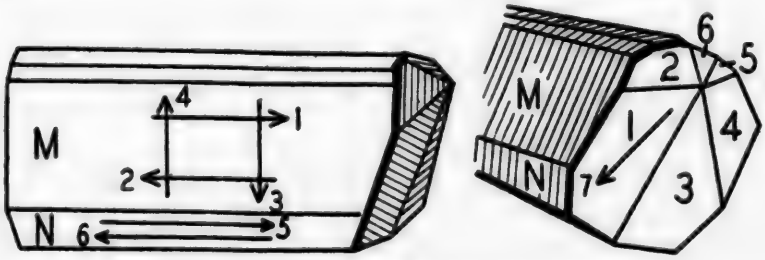

FIG. 5. Crystal 102. Arrows indicate directions of microcuts

On none of the crystal faces could any regular difference in microhardness be noted with varying direction of cutting. Furthermore, the face type did not seem to have any regular relation to the average microhardness. On the five crystals, a total of 638 readings on 82 microcuts gave average widths ranging from 2.8 to 3.0 microns and average microhardnesses ranging from 1110 to 1280. The finding

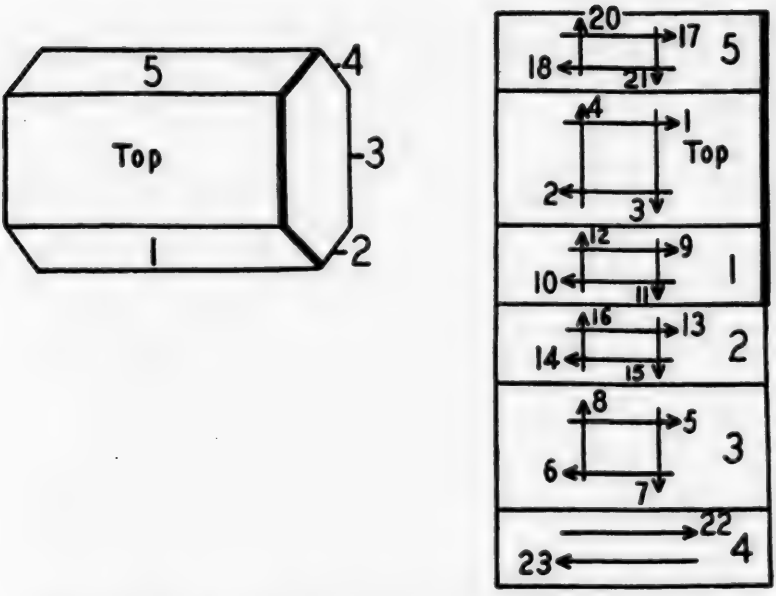

FIG. 6. Crystal 103. Arrows indicate directions of paired microcuts on various faces

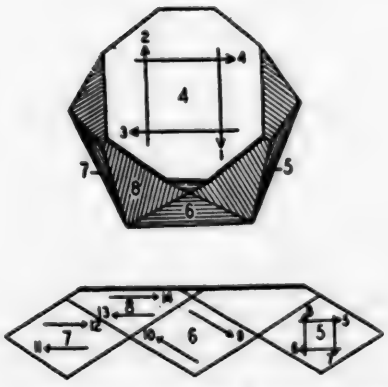

FIG. 7. Crystal 104. From arrows indicating directions of paired microcuts, relations with respect to crystallographic axes may be noted.

TABLE II

CRYSTAL	FACE TYPE	FACE NO.	CUT NO.	NO. READINGS	AV. WIDTH (μ)	AV. MICRO-HARDNESS
100	$10\bar{1}0$	1	1	6	2.8	*1280*
			2	6	2.8	
			3	6	2.8	(1190)
			4	6	2.9	
	0001	"Bottom"	5	10	2.8	*1280*
			6	10	2.8	
			7	10	2.8	
			8	10	2.8	
	0001	"Top"	9	8	2.8	*1280*
			10	8	2.8	
			11	8	2.8	
			12	8	2.8	
	$10\bar{1}0$	2	13	8	2.8	*1280*
			14	8	2.8	
			15	6	2.9	(1190)
			16	6	2.8	
	$10\bar{1}0$	3	17	6	2.8	*1280*
			18	6	2.8	
	Diagonal cut on face "Bottom"			10	2.8	1280
101	$10\bar{1}1$	A	1	10	3.0	1110
			2	10	2.9	1190
			3	10	3.0	1110
			4	10	2.9	1190
	$10\bar{1}1$	B	5	6	2.9	1190
			6	6	3.0	1110
			7	6	3.0	1110
			8	6	2.9	1190
	$10\bar{1}0$	1	9	6	2.9	*1190*
			10	6	2.9	
			11	6	3.0	(1110)
			12	6	2.9	
	$10\bar{1}0$	2	13	6	2.9	(1280)
			14	6	2.8	
			15	6	2.9	*1190*
			16	6	2.9	

TABLE II—*Continued*

CRYSTAL	FACE TYPE	FACE NO.	CUT NO.	NO. READINGS	AV. WIDTH (μ)	AV. MICRO-HARDNESS
101	$10\bar{1}1$	C	17	6	3.0	1110
			18	6	2.9	1190
	$10\bar{1}0$	3	19	6	3.0	1110
			20	6	2.9	1190
102[a]	$10\bar{1}0$	M	1	10	3.0	*1110*
			2	10	2.9	
			3	10	3.0	(1190)
			4	10	3.0	
	$11\bar{2}0$	N	5	10	2.9	*1190*
			6	10	2.9	
	$10\bar{1}1$	1	7	10	3.0	*1110*
103	$10\bar{1}0$	"Top"	1	10	3.0	*1110*
			2	10	3.0	
			3	10	3.0	
			4	10	3.0	
	$10\bar{1}0$	"Bottom"	5	10	3.0	*1110*
			6	10	3.0	
			7	10	3.0	
			8	10	3.0	
	$10\bar{1}0$	1	9	10	3.0	*1110*
			10	10	3.0	
			11	6	2.9	(1190)
			12	6	3.0	
	$10\bar{1}0$	2	13	10	3.0	1110
			14	10	3.0	
			15	6	2.9	(1190)
			16	6	3.0	

[a] Robert D. Butler at the Massachusetts Institute of Technology made goniometer measurements on crystal 102. He reports that in the prism zone faces were 30° apart. Twelve reflections were obtained, only one of which was poor. Therefore first and second order prisms are present. A zone was obtained which included one of the prisms and the pyramid. By goniometer, two pyramid faces make an angle of 80°35′ corresponding to 40°17′ against the basal pinacoid. (Dana's Textbook of Mineralogy, p. 704, cx = 40°18′.)

TABLE II—*Concluded*

CRYSTAL	FACE TYPE	FACE NO.	CUT NO.	NO. READINGS	AV. WIDTH (μ)	AV. MICRO-HARDNESS
103	$10\bar{1}0$	5	17	10	2.9	1190
			18	10	3.0	1110
			20	6	3.0	1110
			21	6	2.9	1190
	$10\bar{1}0$	4	22	10	2.9	1190
			23	10	3.0	1110
104[b]	0001	4	1	10	2.9	*1190*
			2	10	2.9	
			3	10	2.8	(1280)
			4	10	2.9	
	$10\bar{1}0$	5	5	5	2.8	(1280)
			6	5	2.9	
			7	5	2.9	*1190*
			8	5	2.9	
	$10\bar{1}0$	6	9	5	2.8	1280
			10	5	2.9	1190
	$10\bar{1}0$	7	11	5	2.9	1190
			12	5	2.8	1280
	$11\bar{2}1$	8	13	5	2.8	*1280*
			14	5	2.8	

[b] The faces for crystal 104 were identified by Robert D. Butler. By goniometer, the angle between faces 4 and 8 is 55°38′. (Dana's System of Mineralogy, p. 763, cs = 55°45½′.)

(Table I) of the variation in the microhardness of apatite specimens from 870 to 1480 and the average microhardnesses of the "perfect" crystals of 1110 to 1280 is not at all unusual for mineral specimens and may be ascribed to differences in texture or to the presence of impurities. That the microhardness reported here for apatite coincide with the microhardnesses previously found for enamel is noteworthy. The range in microhardness for moist (untreated) enamel is from 300 to 1000 with most average values around 600; values lying within the limits which might be expected of a tissue in which apatite, protein and water are present in approximately the proportions of 98:0.2:2. The

previous findings of certain enamel microhardnesses greater than apatite were not disturbing *per se* because Proell and Schubert (4) have described a similar difference between young enamel, apatite and enamel of old teeth. However, the microhardnesses of apatite reported here as greater than moist enamel seem to give a more consistent description which excludes the necessity for supposing some special hardness of the enamel crystals.

SUMMARY

The average microhardness of 17 specimens of apatite varied from 870 to 1740. On five crystals selected for "perfect" surfaces, the average microhardness ranged from 1110 to 1280.

The authors acknowledge with pleasure the assistance of Professor Q. D. Singewald of the Department of Geology, the University of Rochester.

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